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GEI Consultants

Geotechnical
Environmental and
Water Resources
Engineering

Approval Request for Media Closure Criteria

MacDermid, Inc.

526 Huntingdon Avenue
Waterbury, Connecticut

Submitted to:

Submitted by:
GEI Consultants, Inc.
455 Winding Brook Drive
Glastonbury, CT 06033
860-368-5300

July 2, 2009

Project 073290-*-1001

Frederick W. Johnson, LEP
Senior Vice President

GEI Consultants

Geotechnical
Environmental
Water Resources
Ecological

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Mr. David Ringquist
Bureau of Water Protection and Land Reuse
Department of Environmental Protection
79 Elm Street
Hartford, CT 06106

RE: MacDermid, Inc., 576 Huntingdon Avenue, Waterbury, Connecticut

Dear Mr. ~~Ringquist~~ Dave:

GEI Consultants, Inc. (GEI) has been working with MacDermid to investigate potential site contamination issues under a Stewardship Permit. As part of these activities, GEI is implementing closure of former Resource Recovery and Conservation Act (RCRA) units according to previously-approved closure plans. During closure activities at the former Copper Etchant Recycling Area (RCRA area D), we discovered the presence of certain chemical substances in the floor and associated coatings for which no remediation standards exist. Accordingly, in advance of the final closure report, we submit this request for approval for site-specific media closure criteria.

If you have any questions, please do not hesitate to contact Barry Giroux or me.

Very truly yours,

GEI Consultants, Inc.

Frederick W. Johnson, LEP
Sr. Vice President

FWJ/amm
c: Richard Nave, MacDermid
Carolyn Casey, EPA
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1. Introduction

GEI Consultants, Inc. (GEI) prepared this request for additional media closure criteria (MCC) on behalf of MacDermid, Inc. pursuant to Section 8.2 of the approved Resource Conservation and Recovery Act (RCRA) Closure Plan (regarding closure of its former Copper Etchant Recycling Area). The property is located at 526 Huntingdon Avenue in Waterbury, Connecticut (Site) (Figure 1).

RCRA closure activities were conducted by GEI in November 2008. These closure activities included the sampling the concrete floor in the Copper Etchant Recycling Area, RCRA Area D according to the *Closure Plan Modification for MacDermid Incorporated Hazardous Waste Storage Areas* prepared by Loureiro Engineering Associates, Inc., dated September 2002 as revised on October, 2002, December 2002, and with revisions dated January 24, 2003 and March 7, 2003 ("the Closure Plan").

The investigation detected chloroethane, 2-hexanone, methyl methacrylate, 1,2,2-trichloro 1,1,2-trifluroethane, and benzyl alcohol in low concentrations in the samples taken. Connecticut Department of Environmental Protection (CTDEP) has not established state-wide criteria for either compound within the Remediation Standard Regulations (RSRs).

The approved closure plan requires that criteria be developed for these additional polluting substances, i.e., substances with no promulgated criteria, if they are detected at a Site. The criteria developed must be approved by the CTDEP prior to use. This application proposes Site-specific standards for industrial/commercial direct exposure (RDEC) and groundwater classified "GB" pollutant mobility criteria (GB PMC) for chloroethane, 2-hexanone, methyl methacrylate, 1,2,2-trichloro 1,1,2-trifluroethane, and benzyl alcohol.

Three CTDEP reference documents were used to prepare this document:

- RSRs, Sections 22a-133k-1 through k-3 of the Regulations of Connecticut State Agencies
- Bureau of Water Management Memo, "CT Remediation Standard Regulation-Corrected Criteria Formulas," dated November 18, 2002
- CTDEP "Draft RSR Revisions" dated August 11, 2008

This request contains the information required under the RSRs Section 22a-133 (k) 2 (b) (4) for Additional Polluting Substance Direct Exposure Criteria and 22a-133 (k) 2 (c) (2) for Additional Criteria for PMC in a GB Area.

According to these references and others, this application provides the following information:

- Site Description and History
- Environmental Setting
- Proposed Additional Chloroethane Standards; Industrial/Commercial (I/C) DEC and GB PMC
- Proposed Additional 2-Hexanone Standards; I/C DEC and GB PMC
- Proposed Additional Methyl Methacrylate Standards; I/C DEC and GB PMC
- Proposed Additional 1,2,2-Trichloro-1,1,2-Trifluoroethane Standards; I/C DEC and GB PMC
- Proposed Additional Benzyl Alcohol Standards; I/C DEC and GB PMC

2. Site Description and History

The Site is located at 526 Huntingdon Avenue in Waterbury, Connecticut and includes two parcels of land, the South Parcel and the North Parcel. The South Parcel is approximately 11 acres, is located on the southern side of Huntingdon Avenue, and has three interconnected buildings located on site. The North Parcel is approximately 30 Acres and is located on the northern side of Huntingdon Avenue and is primarily covered with vegetation. The Site location is shown in Figure 1.

MacDermid, Inc. has been in operation at the 526 Huntingdon Avenue in Waterbury, Connecticut location since 1922. Before 1916, the property was owned by the Metal Specialty Company; it is not known what this company produced. From 1916 to 1928, the property was owned by the Waterbury Steel Ball Company (City of Waterbury, 1993). The Waterbury Steel Ball Company leased the property to MacDermid until 1950, when MacDermid purchased the property (City of Waterbury. 1993). MacDermid, Inc. was formerly in the business of blending or compounding of chemical materials used in the metal finishing, plating on plastics and printed circuit industries, as well as the recycling of spent chemicals from other MacDermid facilities and customers. MacDermid ceased operations at the site on December 31, 2003. The facility was permitted as of August 8, 1994, as a commercial hazardous waste storage and recycling facility for RCRA and non-RCRA hazardous wastes. Pursuant to CGS Section 4-182 the existing operating permit (DEP/HWM-151-208) was revoked upon issuance of the Stewardship Permit, which is described below. This facility is currently regulated under RCRA as a generator and a treatment storage and disposal facility (TSDF) of hazardous waste. However, waste generation and handling activities have stopped. MacDermid is in the process of closing the previous RCRA-regulated storage facilities at the Site.

On September 28, 2007 the CTDEP issued MacDermid, Inc. a "Stewardship Permit" for the closure of the Huntingdon Avenue facility. The Stewardship Permit regulates and authorizes MacDermid, Inc. to complete environmental investigation and cleanup ("closure" and "corrective action" measures) in accordance with Connecticut General Statutes (CGS) Sections 22a-6, 22a-449(c) and 22a-454, and Section 22a-449(c)-II of the Regulations of Connecticut State Agencies (RCSA). Because the site was permitted under RCRA for hazardous waste storage and recycling, the Solid Waste Management Units (SWMUs) covered by the RCRA permit must be closed in accordance with the conditions of the Closure Plan submitted and accepted as part of the Part B Permit Renewal application of 1999 (with modifications). Additional areas of Concern identified in the Stewardship Permit must be similarly closed, as well as any additional SWMUs and areas of concern discovered during the course of groundwater monitoring, field investigations, environmental audits, or other means.

The Spent Copper Recycling Area (RCRA Area D) was located in the northwestern portion of the Huntingdon Avenue building in the process area located adjacent to the Copper Etchant Waste Storage Tanks (figure 2). A total of eight aboveground storage tanks, ranging in capacity from 3,200-gallons to 5,300-gallons, comprise the process of copper etchant recycling. This area included two stainless steel reactor storage tanks with capacities of 3,800-gallons and 5,000-gallons. These two tanks represent the RCRA regulated portion of the Spent Copper Recycling area. This regulated unit is used for recycling of spent copper etchant, as described below.

Bulk spent copper etchant was pumped from the Copper Etchant Waste Storage Tanks to one of the two stainless steel reactors, which range in size from 3,800-gallons to 5,000-gallons. A proprietary chemical was added and the solution heated to precipitate copper, which remained in the tank. Ammonia was boiled off through stainless steel piping into one of six receiving fiberglass tanks (3,200-gallons (4), 4,000-gallon (1) and 5,300-gallon (1)), which also contained a proprietary chemical. The reconstituted solution produced a non-copper bearing etchant which was pumped via piping into either three 6,300-gallon storage tanks or directly into tanker trucks for off-site delivery. Copper oxide sludge precipitated during the process was pumped through a filter press and dewatered and stored in fiber drums or tote bags for off-site sale or reused on-site in liquid form to produce copper bearing products.

Secondary containment was provided for the stainless steel RCRA tanks, by an epoxy-coated concrete floor, building walls, floor trenches and collection sumps. The original application date of the epoxy is unknown; however, Stonclad HT was applied to the area in 1994. The epoxy coating cracked, chipped and was worn away in some areas. The floor trenches gravity drain to a transfer sump which then directs wastewater treatment system

3. Environmental Setting

3.1 Groundwater

Groundwater in the Site vicinity is classified by the CTDEP as GB. The Site and vicinity are supplied potable water by the Aquarion Water Company. No public water supply wells or surface water sources are located within one mile of the Site.

3.2 Surface Water

The surface water classification of the Naugatuck River and Steele Brook, located in the vicinity of the facility, are C/B and B, respectively.

3.3 Surficial Soils

The *1992 USGS Surficial Materials Map of Connecticut* depicts surficial materials in the Site vicinity as consisting of alluvium overlying sand. This information is supported by historical and current boring logs completed for soil boring and groundwater monitoring well installations at the site.

3.4 Bedrock

According to the *1967 USGS Bedrock Geologic Map of the Waterbury Quadrangles*, the Site is underlain by the *Hitchcock Lake Member* of the *Hartland Formation,* described as an assemblage of quartz feldspathic granulites and micaceous feldspar-quartz gneisses and schists. Depth to bedrock in the Site vicinity ranges from 3.5 feet on the northern parcel of the site to approximately 40 feet.

4. Proposed Additional Chloroethane Standards I/CDEC and GB PMC

Chloroethane is a colorless gas at room temperature and pressure. It has a characteristically sharp smell. It is a liquid when stored in pressurized containers; however, the liquid evaporates quickly when exposed to room temperatures and pressures. Chloroethane catches fire easily.

Laboratory tests in animals have shown that long-term exposure can cause cancer in mice. It is not known whether it causes cancer in humans. The International Agency for Research on Cancer (IARC) has concluded that chloroethane is not classifiable as to its carcinogenicity in humans.

The Environmental Protection Agency (EPA's) Integrated Risk Information System (IRIS) has published only a risk based reference concentration for inhalation at 10 milligrams per cubic meter (mg/m^3). A reference dose was derived using EPA default exposure factors of 20 cubic meters (m^3) per day respiration rate and 70 kilograms (kg) body weight. The reference dose was calculated at 2.857 milligrams per kilogram per day $(mg/kg\text{-}day)^{-1}$.

This reference dose was inserted into the CTDEP corrected formula for I/C DEC (Table 2). All of the suggested parameters, including the hazard index, were consistent with published values in the RSRs. The result was a RSR I/C DEC value of **16,352,000 mg/kg**. This value is more than the ceiling (1,000 mg/kg) value published in Appendix N of the Draft RSR Revisions (Appendix A); for volatile compounds such as chloroethane, as such the proposed I/C DEC is the ceiling value of 1,000 mg/kg.

The GB PMC was developed based on the CTDEP groundwater protection criteria (GWPC), which also had to be developed. No risk-based values are established for chloroethane in drinking water (i.e. federal drinking water or tap water standards). The GWPC was calculated using the corrected formula, suggested parameters, and a reference dose of 2.857 $(mg/kg\text{-}day)^{-1}$. Using these values the GWPC was 20,000 micrograms per liter (µg/L). The GB PMC was calculated by multiplying the CTDEP GWPC by 20 to convert from liquid (µg/L) to solid units (microgram per kilogram [µg/kg]). This is a conservative assumption based on the dilution made in a standard leaching test. This value was then multiplied by 10 to account for the GB classification of the site. The GB PMC for chloroethane was calculated at 4,000,000 µg/kg or **4,000 mg/kg.** Calculations for I/C DEC, GWPC, GB PMC values and IRIS information for chloroethane are included in Appendix B.

5. Proposed Additional 2-Hexanone Standards I/C DEC and GB PMC

2-Hexanone is also known as methyl n-butyl ketone, MBK, or propyl acetone. It is a clear, colorless liquid with a sharp odor. It dissolves very easily in water, and can evaporate easily into the air.

The U.S. Department of Health and Human Services has not classified 2-hexanone as to human carcinogenicity. Also, the International Agency for Research on Cancer and the EPA has not classified 2-hexanone as to human carcinogenicity. There is no information available on the potential carcinogenic effects of 2-hexanone in people or in experimental animals. There is no evidence that 2-hexanone causes cancer.

CTDEP has not promulgated any RSR criteria for 2-hexanone. No federal standards (i.e. IRIS) are established the compound. The Superfund Technical Support Center published reference dose of 0.04 $(mg/kg\text{-}day)^{-1}$. This reference dose was inserted into the CTDEP corrected formula for I/C DEC (Table 2). All of the suggested parameters, including the hazard index, were consistent with published values in the RSRs. The result was an RSR I/C DEC value of **228,928 mg/kg**. This value is more than the ceiling value (1,000 mg/kg) published in Appendix N of the Draft RSR Revisions (Appendix A); for volatile compounds such as 2-hexanone, as such the proposed I/C DEC is the ceiling value of 1,000 mg/kg.

The GB PMC was calculated by using the CTDEP GWPC, which also had to be developed. No risk-based values are established for 2-hexanone in drinking water (i.e. federal drinking water or tap water standards). The GWPC was calculated using the corrected formula, suggested parameters, and a reference dose of 0.04 $(mg/kg\text{-}day)^{-1}$. Using these values the GWPC was 280 µg/L. The GB PMC was calculated by multiplying the CTDEP GWPC by 20 to convert from liquid (µg/L) to solid units (µg/kg). This is a conservative assumption based on the dilution made in a standard leaching test. This value was them multiplied by 10 to account for the GB classification of the site. The GB PMC for 2-hexanone was calculated at 56,000 µg/kg or **56 mg/kg.** Calculations for I/C DEC, GWPC, and GB PMC values for 2-hexanone are included in Appendix C.

6. Proposed Additional Methyl Methacrylate Standards I/C DEC and GB PMC

Methyl methacrylate is used in the manufacture of resins and plastics. Methyl methacrylate is irritating to the skin, eyes, and mucous membranes in humans. EPA considers methyl methacrylate not likely to be carcinogenic to humans.

The EPA's IRIS has published a reference dose at 1.4 $(mg/kg\text{-}day)^{-1}$. This reference dose was inserted into the CTDEP corrected formula for I/C DEC (Table 2). All of the suggested parameters, including the hazard index, were consistent with published values in the RSRs. The result was an RSR I/C DEC value of **8,012,408 mg/kg**. This value is more than the ceiling value (1,000 mg/kg) published in Appendix N of the Draft RSR Revisions (Appendix A), for volatile compounds such as methyl methacrylate. As such, the proposed I/C DEC is the ceiling value of 1,000 mg/kg.

The GB PMC was developed based on the CTDEP GWPC, which also had to be developed. No risk-based values are established for methyl methacrylate in drinking water (i.e., federal drinking water or tap water standards). The GWPC was calculated using the corrected formula, suggested parameters, and a reference dose of 1.4 $(mg/kg\text{-}day)^{-1}$. Using these values the GWPC is 9,800 µg/L. The GB PMC was calculated by multiplying the CTDEP GWPC times 20 to convert from liquid (µg/L) to solid units (µg/kg). This is a conservative assumption based on the dilution made in performing a standard leaching test. This value was multiplied then by 10 to GB classification of the Site. GB PMC for methyl methacrylate was calculated at 1,960,000 µg/kg or **1,960 mg/kg.** Calculations for I/C DEC, GWPC, GB PMC values and IRIS information for methyl methacrylate is included in Appendix D.

7. Proposed Additional 1,2,2-Trichloro-1,1,2-Trifluorethane Standards I/C DEC and GB PMC

1,2,2-Trichloro-1,1,2-Trifluoroethane is a colorless man made liquid or gas, it was used in the as a refrigerant and a solvent for degreasing or dry cleaning. EPA considers 1,2,2-Trichloro-1,1,2-Trifluoroethane not likely to be carcinogenic to humans.

The EPA's IRIS has published reference dose at 3.0 $(mg/kg\text{-}day)^{-1}$. This reference dose was inserted into the CTDEP corrected formula for I/C DEC (Table 2). All of the suggested parameters, including the hazard index, were consistent with published values in the RSRs. The result was an RSR I/C DEC value of **17,169,600 mg/kg**. This value is more that the ceiling value (1,000 mg/kg) published in Appendix N of the Draft RSR revisions (Appendix A), for volatile compounds, as such 1,2,2-Trichloro-1,1,2-Trifluoroethane, as such the proposed I/C DEC is the ceiling value of 1,000 mg/kg.

The GB PMC was developed based on the CTDEP GWPC, which also had to be developed. No risk-based values are established for 1,2,2-Trichloro-1,1,2-Trifluoroethane in drinking water (i.e., federal drinking water or tap water standards). The GWPC was calculated using the corrected formula, suggested parameters, and a reference dose of 3.0 $(mg/kg\text{-}day)^{-1}$. The GB PMC was calculated by multiplying the CTDEP GWPC by 20 to convert form liquid (ug/L) to solid units (µg/L). This is a conservative assumption based on the dilution made in a standard leaching test. This value was then multiplied by 10 to account for the GB classification of the site. The GB PMC for 1,2,2-Trichloro-1,1,2-Trifluoroethane was calculated at 4,200,000 µg/kg or **4,200 mg/kg.** Calculations for I/C DEC, GWPC, GB PMC values and IRIS information for 1,2,2-Trichloro-1,1,2-Trifluoroethane are included in Appendix E.

8. Proposed Additional Benzyl Alcohol Standards I/C DEC and GB PMC

Benzyl alcohol is a colorless liquid. It was used as a solvent for inks, paints, lacquers and epoxy resin coatings. The EPA has not classified benzyl alcohol as to human carcinogenicity.

CTDEP has not promulgated any RSR criteria for benzyl alcohol. The EPA's Superfund Provisional Peer Reviewed Toxicity (PPRTV) has published a reference dose at 0.5 (mg/kg-day)[1]. This reference dose was inserted into the CTDEP corrected formula for I/C DEC (Table 2). All of the suggested parameters, including the hazard index, were consistent with published values in the RSRs. The result was an RSR I/C DEC value of **2,861,600 mg/kg**. Since benzyl alcohol is a semi-volatile compound, this value is more than the ceiling value (2,500 mg/kg) published in Appendix N of the Draft RSR Revisions (Appendix A).

The GB PMC was developed based on the CTDEP GWPC, which also had to be developed. No risk-based values are established for benzyl alcohol in drinking water (i.e., federal drinking water or tap water standards). The GWPC was calculated using the corrected formula, suggested parameters, and a reference dose of 0.5 $(mg/kg\text{-}day)^{-1}$. Using these values the GWPC was 3,500 µg/L. The GB PMC was calculated by multiplying the CTDEP GWPC by 20 to convert from liquid (µg/L) to solid (µg/kg). This is a conservative assumption based on the dilution mad in a standard leaching test. This value was multiplied by 10 to account for the GB classification of the site. The GB PMC for benzyl alcohol was calculated at 700,000 µg/kg or **700 mg/kg.** Calculations for I/C DEC, GWPC and GB PMC values and the Superfund PPRTV for benzyl alcohol are included in Appendix F.

9. Conclusions

The table below provides the relevant statistics and proposed additional RSR criteria.

Description	Chloroethane	2-Hexanone	Metyl Methacrylate	1,2,2-Trichloro-1,1,2-Trifluorethane	Benzyl Alcohol
Number of Detections on Site (mg/kg)	1 of 18 samples	1 of 18 samples	1 of 18 samples	1 of 18 samples	11 of 18 samples
Highest Concentration Detected (mg/kg)	.0085	.0066	190	.00084	470
Reporting Limit (mg/kg)	.0012	.0027	1.9	.00071	.19
Proposed I/C DEC (mg/kg)	1,000	1,000	1,000	1,000	2,500
Proposed GB PMC (mg/kg)	4,000	56	1,960	4,200	700

The laboratory reporting limit provided above is based on actual values provided by TestAmerica, Inc. during the RCRA investigation. These reporting limits are orders of magnitude below all of the proposed additional criteria.

MacDermid requests that CTDEP approved I/C DEC for chloroethane, 2-hexanone, methyl methacrylate, and 1,2,2-trichloro-1,1,2-trifluroethane at 1,000 mg/kg and benzyl alcohol at 2,500 mg/kg. MacDermid also requests a GB PMC of 4,000 mg/kg for chloroethane, 56 for mg/kg 2-hexanone, 1,960 mg/kg methyl methacrylate, 4,200 mg/kg 1,2,2-trichloro-1,1,2-trifluroethane, and 700 mg/kg benzyl alcohol. These additional standards are higher than any detections analyzed from the RCRA Closure activities. In our opinion, these criteria will be protective of human health and the environment and not affect the current use of the Site soils, groundwater, or nearby surface water bodies.

Table

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Table 1
Soil Sampling Analytical Results Summary
MacDermid, Inc.

Sample Name: / Sample Date:	RES DEC	I/C DEC	GB PMC	SCER-1 8/8/2008	SCER-2 8/8/2008	SCER-3 8/8/2008	SCER-4 8/8/2008	SCER-5 8/8/2008	SCER-6 8/8/2008	SCER-7 8/8/2008	SCER-8 8/8/2008	SCER-9 8/8/2008	SCER-10 8/8/2008	SCER-11 8/8/2008	SCER-12 8/8/2008	SCER-13 8/8/2008	SCER-14 8/8/2008	SCER-15 8/8/2008	SCER-16 8/8/2008	SCER-17 8/8/2008	SCER-18 8/8/2008
VOCs (mg/kg)																					
Acetone	500	1,000	140	0.029 UJ	0.0044 UJ	**0.18 J**	0.26 J	R	**0.056 J**	**0.53 J**	**1.1 J**	0.03 UJ	**0.12 J**	0.026 UJ	0.03 UJ	**0.18 J**	0.027 UJ	0.019 UJ	**2 J**	**0.26 J**	**0.3 J**
Benzene	21	200	0.2	0.0044 UJ	0.0047 UJ	**0.02 J**	**0.93 J**	R	R	**0.015 J**	**0.004 J**	R	**0.23 J**	R	0.35 U	**0.18 J**	R	R	**0.77 J**	**0.31 J**	**0.018 J**
Butanone, 2-	50	1,000	80	0.0087 UJ	0.0094 UJ	**0.024 J**	**0.053 J**	R	R	4.4 U	**6.3 J**	**0.12 J**	**0.037 J**	R	0.35 U	**0.029 J**	R	R	**0.08 J**	**0.043 J**	**0.055 J**
Butylbenzene, n-	500	1,000	14	0.0044 UJ	0.0047 UJ	4.5 U	4.3 UJ	**0.0018 J**	R	4.4 U	44 UJ	R	1.1 U	R	**0.073 J**	**0.39 J**	R	R	4.2 U	2.2 U	5.4 U
Butylbenzene,sec-	500	1,000	14	0.0044 UJ	0.0047 UJ	**0.0012 J**	4.3 U	R	R	4.4 U	**0.0043 J**	R	**0.18 J**	**0.0017 J**	0.35 U	**0.23 J**	R	R	**0.0016 J**	2.2 U	**0.0018 J**
Carbon disulfide	500	1,000	140	0.0044 UJ	0.0047 UJ	**0.0011 J**	**0.00068 J**	R	**0.0015 J**	4.4 U	44 U	R	**0.0023 J**	R	0.35 U	**0.00087 J**	R	**0.00076 J**	**0.0014 J**	2.2 U	5.4 U
Chloroethane	SS-210	NE	NE	0.0044 UJ	0.0047 UJ	4.5 U	4.3 U	R	R	4.4 U	**0.0085 J**	R	1.1 U	R	0.35 U	1.2 U	R	R	4.2 U	2.2 U	5.4 U
Chloromethane	47	440	0.54	0.0044 UJ	0.0047 UJ	4.5 U	4.3 U	R	R	4.4 U	44 U	R	1.1 U	R	0.35 U	1.2 U	R	R	**0.001 J**	2.2 U	5.4 U
Dichloroethane,1,1-	500	1,000	14	0.0044 UJ	0.0047 UJ	0.008 J	4.3 U	R	R	4.4 U	44 U	R	1.1 U	R	0.35 U	1.2 U	R	R	4.2 U	2.2 U	5.4 U
Ethylbenzene	500	1,000	10.1	**0.0069 J**	0.0047 UJ	**2.3 J**	**2.3 J**	**0.0091 J**	R	**1.2 J**	**5.5 J**	**0.0027 J**	**2.5**	**0.0059 J**	**0.0067 J**	**0.74 J**	**0.0018 J**	R	**2.1 J**	**0.71 J**	**1.2 J**
Hexanone,2-	NE	NE	NE	0.0087 UJ	0.0094 UJ	4.5 U	**0.0066 J**	R	R	4.4 U	44 U	R	1.1 U	R	0.35 U	1.2 U	R	R	4.2 U	2.2 U	5.4 U
Isopropyl benzene	500	1,000	132	**0.002 J**	0.0047 UJ	**36**	**44**	**0.013 J**	**0.0055 J**	**14**	**0.1 J**	**0.0024 J**	**41**	**0.0066 J**	**0.13 J**	**7.5**	**0.0015 J**	R	**81**	**16**	**15**
Isopropyltoluene,4-	500	1,000	41.8	**0.0011 J**	0.0047 UJ	4.5 U	4.3 U	**0.0034 J**	R	4.4 U	44 U	R	1.1 U	**0.0035 J**	**0.0042 J**	1.2 U	R	R	4.2 U	2.2 U	5.4 U
Methyl methacrylate	NE	NE	NE	R	R	**160 J**	**260 J**	R	R	**60 J**	**1700 J**	R	**190 J**	R	R	**83 J**	R	R	**310 J**	**120 J**	**270 J**
Methyl Isobutyl ketone (MIBK)	500	1,000	14	0.0044 UJ	0.0047 UJ	4.5 U	4.3 U	**0.16 J**	R	4.4 U	**0.01 J**	**0.015 J**	**0.0077 J**	**0.0087 J**	**0.6**	1.2 U	**0.022 J**	**0.0051 J**	**0.0061 J**	2.2 U	**0.0054 J**
Methylene chloride	82	760	1	0.017 UJ	0.019 UJ	0.018 UJ	4.3 U	R	R	4.4 U	44 U	R	1.1 U	R	0.35 U	1.2 U	0.019 UJ	R	4.2 U	2.2 U	5.4 U
Naphthalene	1,000	2,500	56	**0.0055 J**	0.0047 UJ	**12**	**0.0041 J**	**0.0018 J**	R	**0.018 J**	**0.0055 J**	**0.0025 J**	1.1 U	**0.0015 J**	**0.88 J**	**1 J**	R	R	**0.0031 J**	2.2 UJ	5.4 UJ
Propylbenzene, n-	500	1,000	14	**0.0051 J**	0.0047 UJ	**1.6 J**	**1.8 J**	**0.02 J**	R	**0.64 J**	**0.075 J**	R	**1.3**	**0.011 J**	**0.26 J**	**0.52 J**	R	R	**2.6 J**	**0.61 J**	**1 J**
Styrene	500	1,000	20	**0.0041 J**	0.0047 UJ	**160**	**110**	**0.014 J**	**0.023 J**	**130**	**660**	**0.082 J**	**38 J**	**0.05 J**	**0.013 J**	**36**	**0.012 J**	**0.0039 J**	**94**	**48**	**110**
Tetrachloroethene	12	110	1	0.0044 UJ	0.0047 UJ	4.5 U	4.3 U	R	R	4.4 U	**0.0032 J**	R	1.1 U	R	0.35 U	1.2 U	R	R	4.2 U	**0.00074 J**	5.4 U
Toluene	500	1,000	67	0.0044 UJ	0.0047 UJ	0.0045 UJ	**0.0056 J**	**0.0043 J**	R	**0.0043 J**	**0.0085 J**	R	1.1 U	**0.039 J**	**0.0038 J**	1.2 U	R	R	**0.0044 J**	2.2 U	5.4 U
Trichloro-1,2,2-trifluoroethane, 1,1,2-	NE	NE	NE	0.0044 UJ	0.0047 UJ	**0.00084 J**	4.3 U	R	R	4.4 U	44 U	R	1.1 U	R	0.35 U	1.2 U	R	R	4.2 U	2.2 U	5.4 U
Trichlorobenzene, 1,2,4-	680	2,500	14	0.0044 UJ	0.0047 UJ	4.5 U	4.3 UJ	R	R	4.4 UJ	44 UJ	R	1.1 U	R	0.35 UJ		R	R	4.2 UJ	**0.0032 J**	5.4 UJ
Trichloroethene	56	520	1	0.0044 UJ	0.0047 UJ	4.5 U	4.3 U	R	R	4.4 U	44 U	R	1.1 U	R	0.35 U	1.2 U	R	R	4.2 U	2.2 U	5.4 U
Trimethylbenzene, 1,2,4-	500	1,000	70	**0.057 J**	0.0047 UJ	**0.0013 J**	4.3 U	**0.18 J**	**0.0022 J**	**0.0093 J**	**0.023 J**	**0.056 J**	**3.7**	**0.13 J**	**2.9**	**0.0015 J**	**0.02 J**	**0.0015 J**	**5.7**	2.2 U	**0.0016 J**
Trimethylbenzene, 1,3,5-	500	1,000	70	**0.015 J**	0.0047 UJ	4.5 U	4.3 U	**0.042 J**	R	**0.0047 J**	**0.009 J**	**0.0082 J**	**1.8**	**0.035 J**	**0.95**	1.2 U	**0.0076 J**	R	**3.5 J**	2.2 U	**0.00097 J**
Xylene, m,p-	NE	NE	NE	**0.027 J**	0.0047 UJ	**1.4 J**	**0.0095 J**	**0.037 J**	R	**0.02 J**	**0.21 J**	**0.018 J**	**3.3**	**0.025 J**	**0.16 J**	**0.0038 J**	**0.011 J**	R	**0.0064 J**	**0.0027 J**	**0.0052 J**
Xylene, o-	NE	NE	NE	**0.0085 J**	0.0047 UJ	**0.01 J**	**0.0079 J**	**0.017 J**	R	**0.0089 J**	**0.2 J**	**0.011 J**	**1.3**	**0.013 J**	**0.22 J**	**0.0018 J**	**0.0081 J**	R	**1.1 J**	**0.0027 J**	**0.0053 J**
Total Xylene	500	1,000	19.5	**0.0355**	ND	**1.41**	**0.0174**	**0.054**	ND	**0.0289**	**0.41**	**0.029**	**4.6**	**0.038**	**0.38**	**0.0056**	**0.0191**	ND	**1.1064**	**0.0054**	**0.0105**
SVOCs (mg/kg)																					
Acenaphthylene	1,000	2,500	84	1.4 U	0.69 U	1.4 U	0.34 U	1.4 U	1.4 U	1.4 UJ	1.4 U	1.4 U	1.3 U	1.4 U	1.4 U	1.3 U	1.7 U	1.4 U	1.3 U	2.7 U	2.7 U
Anthracene	1,000	2,500	400	1.4 U	0.69 U	1.4 U	0.34 U	1.4 U	1.4 U	1.4 UJ	1.4 U	1.4 U	1.3 U	1.4 U	1.4 U	1.3 U	1.7 U	1.4 U	1.3 U	2.7 U	2.7 U
Benz[a]anthracene	1	7.8	1	1.4 U	0.69 U	1.4 U	0.34 U	1.4 U	1.4 U	1.4 UJ	1.4 U	1.4 U	1.3 U	1.4 U	1.4 U	1.3 U	1.7 U	1.4 U	1.3 U	2.7 U	2.7 U
Benzo[a]pyrene	1	1	1	1.4 U	0.69 U	1.4 U	0.34 U	1.4 U	1.4 U	1.4 UJ	1.4 U	1.4 U	1.3 U	1.4 U	1.4 U	1.3 U	1.7 U	1.4 U	1.3 U	2.7 U	2.7 U
Benzo[b]fluoranthene	1	7.8	1	1.4 U	0.69 U	1.4 U	0.34 U	1.4 U	1.4 U	1.4 UJ	1.4 U	1.4 U	1.3 U	1.4 U	1.4 U	1.3 U	1.7 U	1.4 U	1.3 U	2.7 U	2.7 U
Benzo[g,h,i]perylene	1,000	2,500	42	1.4 U	0.69 U	1.4 U	0.34 U	1.4 U	1.4 U	1.4 UJ	1.4 U	1.4 U	1.3 U	1.4 U	1.4 U	1.3 U	1.7 U	1.4 U	1.3 U	2.7 U	2.7 U
Benzo[k]fluoranthene	8.4	78	1	1.4 U	0.69 U	1.4 U	0.34 U	1.4 U	1.4 U	1.4 UJ	1.4 U	1.4 U	1.3 U	1.4 U	1.4 U	1.3 U	1.7 U	1.4 U	1.3 U	2.7 U	2.7 U
Benzyl alcohol	NE	NE	NE	**190**	0.69 U	1.4 U	**0.37**	**260**	1.4 U	**12 J**	**0.86 J**	**2.3**	**0.34 J**	**74**	**520**	1.3 U	**4.6**	**11**	1.3 U	2.7 U	2.7 U
Bis(2-ethylhexyl)phthalate	44	410	11	**0.87 J**	**0.21 J**	**0.68 J**	**0.66**	1.4 U	**0.77 J**	**0.48 J**	**2.2**	**0.8 J**	**1.7**	1.4 U	1.4 U	**0.56 J**	**0.93 J**	1.4 U	**1.6**	**0.98 J**	**1.2 J**
Chrysene	84	780	1	1.4 U	0.69 U	1.4 U	0.34 U	1.4 U	1.4 U	1.4 UJ	1.4 U	1.4 U	1.3 U	1.4 U	1.4 U	1.3 U	1.7 U	1.4 U	1.3 U	2.7 U	2.7 U
Dibenz[a,h]anthracene	1	1	1	1.4 U	0.69 U	1.4 U	0.34 U	1.4 U	1.4 U	1.4 UJ	1.4 U	1.4 U	1.3 U	1.4 U	1.4 U	1.3 U	1.7 U	1.4 U	1.3 U	2.7 U	2.7 U
Dimethyl phthalate	1,000	2,500	1,100	1.4 U	0.69 U	1.4 U	**0.19 J**	1.4 U	1.4 U	1.4 UJ	1.4 U	1.4 U	1.3 U	**0.47 J**	1.4 U	1.3 U	1.7 U	1.4 U	1.3 U	2.7 U	2.7 U
Dimethylphenol, 2,4-	1,000	2,500	28	1.4 UJ	0.69 U	1.4 UJ	0.34 U	1.4 UJ	1.4 UJ	1.4 UJ	1.4 UJ	1.4 UJ	1.3 U	1.4 UJ	1.4 UJ	1.3 U	1.7 UJ	1.4 UJ	1.3 U	2.7 UJ	**11 J**
Di-n-butyl phthalate	1,000	2,500	140	1.4 U	0.69 U	1.4 U	0.34 U	1.4 U	1.4 U	1.4 UJ	1.4 U	1.4 U	1.3 U	1.4 U	1.4 U	1.3 U	1.7 U	1.4 U	1.3 U	2.7 U	2.7 U
Di-n-octyl phthalate	1,000	2,500	20	1.4 U	0.69 U	1.4 U	**0.22 J**	1.4 U	1.4 U	1.4 UJ	**0.22 J**	1.4 U	1.3 U	1.4 U	1.4 U	**0.33 J**	1.7 U	1.4 U	1.3 U	2.7 U	2.7 U
Fluoranthene	1,000	2,500	56	1.4 U	**0.2 J**	1.4 U	0.34 U	1.4 U	1.4 U	**0.29 J**	1.4 U	1.4 U	1.3 U	1.4 U	1.4 U	1.3 U	1.7 U	1.4 U	1.3 U	2.7 U	2.7 U
Indeno[1,2,3-cd]pyrene	1	7.8	1	1.4 U	0.69 U	1.4 U	0.34 U	1.4 U	1.4 U	1.4 UJ	1.4 U	1.4 U	1.3 U	1.4 U	1.4 U	1.3 U	1.7 U	1.4 U	1.3 U	2.7 U	2.7 U
Methylnaphthalene,2-	474	2,500	9.8	**0.82 J**	0.69 U	1.4 U	0.34 U	1.4 U	1.4 U	1.4 UJ	1.4 U	1.4 U	1.3 U	1.4 U	1.4 U	1.3 U	1.7 U	1.4 U	1.3 U	2.7 U	2.7 U
Methylphenol,2-	1,000	2,500	70	1.4 UJ	0.69 U	1.4 UJ	0.34 U	1.4 UJ	1.4 UJ	1.4 UJ	**4.8 J**	1.4 UJ	1.3 U	1.4 UJ	1.4 UJ	1.3 U	1.7 UJ	1.4 UJ	1.3 U	2.7 UJ	2.7 UJ
Naphthalene	1,000	2,500	56	**0.3 J**	0.69 U	**0.3 J**	0.34 U	1.4 U	1.4 U	**0.66 J**	1.4 U	1.4 U	1.3 U	1.4 U	1.4 U	**0.38 J**	1.7 U	1.4 U	1.3 U	2.7 U	2.7 U
Phenanthrene	1,000	2,500	40	1.4 U	**0.33 J**	1.4 U	0.34 U	1.4 U	1.4 U	**0.4 J**	1.4 U	1.4 U	1.3 U	1.4 U	1.4 U	1.3 U	1.7 U	1.4 U	1.3 U	2.7 U	2.7 U
Phenol	1,000	2,500	800	**0.63 J**	0.69 U	1.4 UJ	0.34 U	**9.5 J**	**1.1 J**	1.4 UJ	**220**	1.4 UJ	**1 J**	**19 J**	**30 J**	**1.8**	1.7 UJ	**13 J**	**2.5**	2.7 UJ	2.7 UJ
Pyrene	1,000	2,500	40	1.4 U	**0.15 J**	1.4 U	0.34 U	1.4 U	1.4 U	1.4 UJ	1.4 U	1.4 U	1.3 U	1.4 U	1.4 U	1.3 U	1.7 U	1.4 U	1.3 U	2.7 U	2.7 U
Trichlorobenzene, 1,2,4-	680	2,500	14	1.4 U	0.69 U	1.4 U	0.34 U	1.4 U	1.4 U	1.4 UJ	1.4 U	1.4 U	1.3 U	1.4 U	1.4 U	1.3 U	1.7 U	1.4 U	1.3 U	2.7 U	2.7 U
Total Metals (mg/kg)																					
Antimony	27	8,200	NA	12.9 U	**11 J**	13.2 U	12.9 U	12.9 U	12.9 U	13.3 U	12.9 U	12.9 U	12.7 U	13 U	12.9 U	12.8 U	12.9 U	12.9 U	12.7 U	12.8 U	12.9 U
Arsenic	10	10	NA	**2.5 J**	**11.1**	**3.4 J**	**3.9 J**	**2.9 J**	**2.4 J**	**3.2 J**	**3 J**	**2.3 J**	**4.8**	**2.6 J**	**2.3 J**	**5**	**3.2 J**	**3.1 J**	**4.2**	**5.5**	**4.8**
Barium	4,700	140,000	NA	**62.5 J**	**99.1 J**	**46.8 J**	**103 J**	**70.8 J**	**119 J**	**123 J**	**82 J**	**159 J**	**117 J**	**71.2 J**	**64.1 J**	**114 J**	**87.3 J**	**40.1 J**	**110 J**	**128 J**	**117 J**
Beryllium	2	2	NA	**0.48 J**	**0.45 J**	**0.84 J**	**0.5 J**	**0.55 J**	**0.61 J**	**0.47 J**	**0.41 J**	**0.66 J**	**0.45 J**	**0.54 J**	**0.44 J**	**0.49 J**	**0.67 J**	**0.72 J**	**0.45 J**	**0.64 J**	**0.49 J**
Cadmium	34	1,000	NA	6.5 U	**0.91 J**	6.6 U	6.5 U	6.5 U	6.4 U	6.6 U	6.4 U	6.5 U	6.3 U	6.5 U	6.5 U	6.4 U	6.5 U	6.5 U	6.3 U	6.4 U	6.4 U
Chromium	100/3,900	100/51,00	NA	**17**	**50.4**	**76**	**21.1**	**22.2**	**30.8**	**134**	**36.1**	**15**	**44.7**	**19.1**	**12.3**	**23**	**15**	**15.4**	**55.7**	**22.2**	**25.3**
Cobalt	70	NE	NA	**4.7 J**	**6.5 J**	**21.6**	**16.4**	**4.7 J**	**6.9 J**	**25.8**	**18.5**	**4.4 J**	**11.6 J**	**5 J**	**4.2 J**	**17**	**4.4 J**	**8.7 J**	**16.3**	**12 J**	**16.1**
Copper	2,500	76,000	NA	**321 J**	**9310 J**	**1640 J**	**800 J**	**77.4 J**	**856 J**	**87.7 J**	**6040 J**	**152 J**	**1580 J**	**54.8 J**	**97.1 J**	**4580 J**	**83.3 J**	**350 J**	**333 J**	**240 J**	**101 J**
Lead	400	1,000	NA	**2.6 J**	**32**	**48.6**	**9.9**	**2.5 J**	**7.1**	**71.5**	**53.7**	**2.9**	**11.6**	**2.1 J**	**2 J**	**12.7**	**2.6**	**2.6**	**5.1**	**8.4**	**8.9**
Mercury	20	610	NA	**0.018**	**0.26**	**0.23**	**0.12**	**0.052**	**0.031**	**0.051**	**1.7**	**0.052**	**0.26**	**0.049**	**0.052**	**0.098**	**0.049**	**0.051**	**0.12**	**0.16**	**0.21**
Nickel	1,400	7500	NA	**13.1**	**500**	**93.1**	**859**	**14.9**	**25.7**	**71.9**	**71.4**	**10.9**	**48.6**	**13.4**	**9.5 J**	**25.9**	**10.9**	**13.3**	**25.5**	**28.7**	**62.5**
Selenium	340	10,000	NA	12.9 U	13 U	13.2 U	12.9 U	12.9 U	12.9 U	13.3 U	12.9 U	12.9 U	12.7 U	13 U	12.9 U	12.8 U	12.9 U	**1.3 J**	12.7 U	12.8 U	12.9 U
Silver	340	10,000	NA	3.9 U	**1.9 J**	**0.52 J**	3.9 U	3.9 U	3.9 U	**8.7**	**1.8 J**	3.9 U	**0.97 J**	3.9 U	3.9 U	**0.48 J**	3.9 U	3.9 U	**0.57 J**	3.8 U	**0.66 J**
Vanadium	470	14,000	NA	**23.8**	**24.5**	**40.7**	**25.2**	**27.6**	**31**	**18.1**	**18.4**	**20**	**25.3**	**25.4**	**18.9**	**28**	**19.5**	**54.4**	**27**	**28.9**	**27.1**
Zinc	20,000	610,000	NA	**26.2**	**105**	**183**	**52.2**	**24.8**	**51.9**	**25.1**	**161**	**23.4**	**72.6**	**31.8**	**21.9**	**51.1**	**22.1**	**35.5**	**37.4**	**43.3**	**36.3**

Table 1
Soil Sampling Analytical Results Summary
MacDermid, Inc.

Notes:

mg/kg - milligrams/kilogram or parts per million (ppm)
BTEX - benzene, toluene, ethylbenzene, and xylenes
VOCs - volatile organic compounds
SVOCs - semivolatile organic compounds

Res DEC - Residential direct exposure criteria means the concentrations identified as residential direct exposure criteria in Appendix A to sections 22a-133k-1 through 22a-133k-3 of the Regulations of Connecticut State Agencies.

I/C DEC - Industrial/commercial direct exposure criteria means the concentrations identified as industrial/commercial direct exposure criteria in Appendix A to sections 22a-133k-1 through 22a-133k-3 of the Regulations of Connecticut State Agencies.

GB - means an area where the ground-water classification is GB

PMC - Pollutant mobility criteria means the concentrations identified in Appendix B to sections 22a-133k-1through 22a-133k-3 of the Regulations of Connecticut State Agencies or any alternative pollutant mobility criteria approved by the Commissioner pursuant to subsection 22a-133k-2(d) of the Regulations of Connecticut State Agencies.

NE - not established

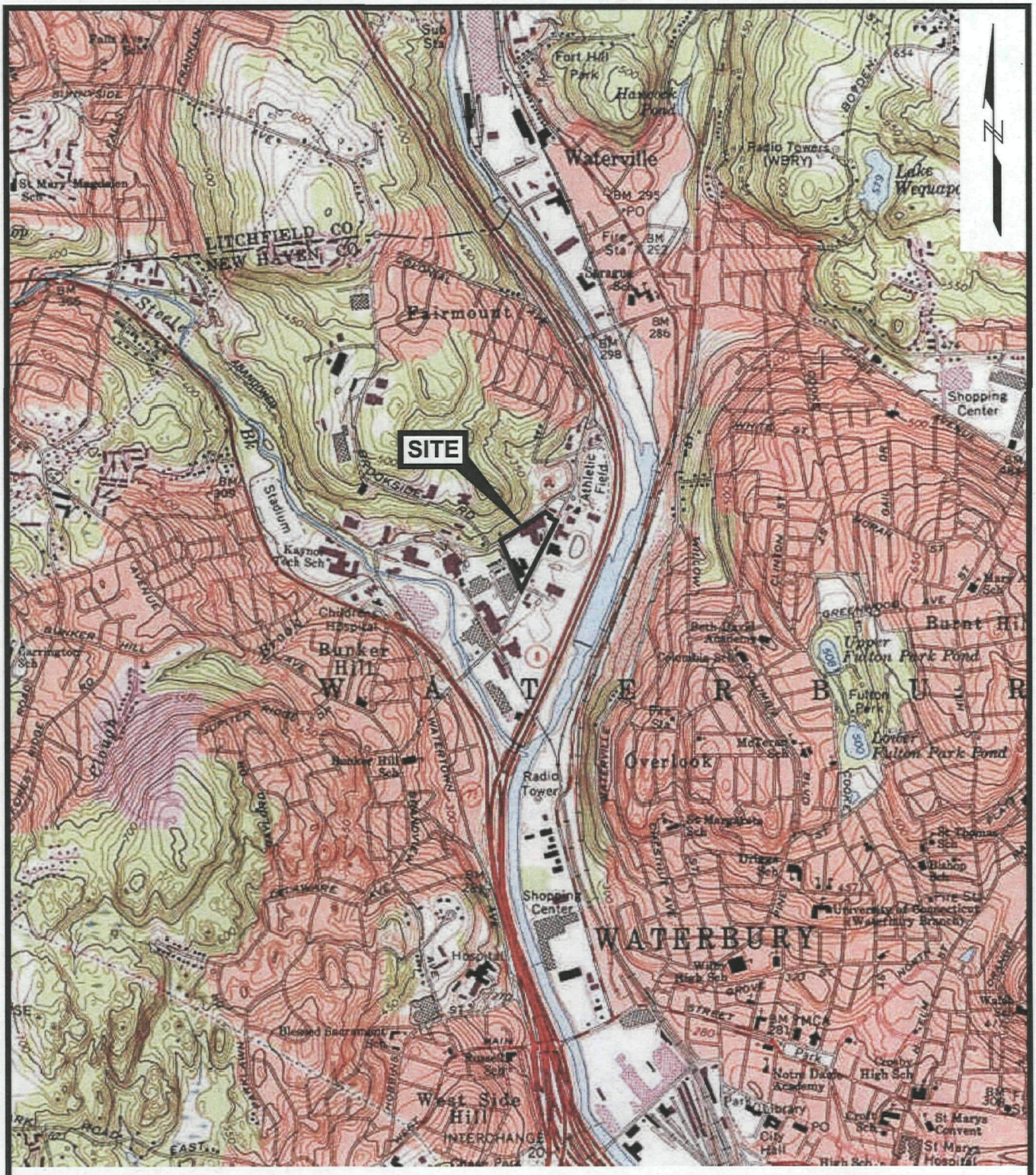
SS - if statewide criteria have not been established, but site specific criteria are available, this is denoted by the prefix "SS" and the most conservative site specific value are listed.

Bolding indicates a detected result value
Shading and bolding indicates that the detected result value exceeds the Remediation Standard it was compared to

Validation Qualifiers:

J - estimated value
U - indicates not detected to the reporting limit for organic analysis and the method detection limit for inorganic analysis
UJ - not detected at or above the reporting limit shown and the reporting limit is estimated
R - rejected

Figures



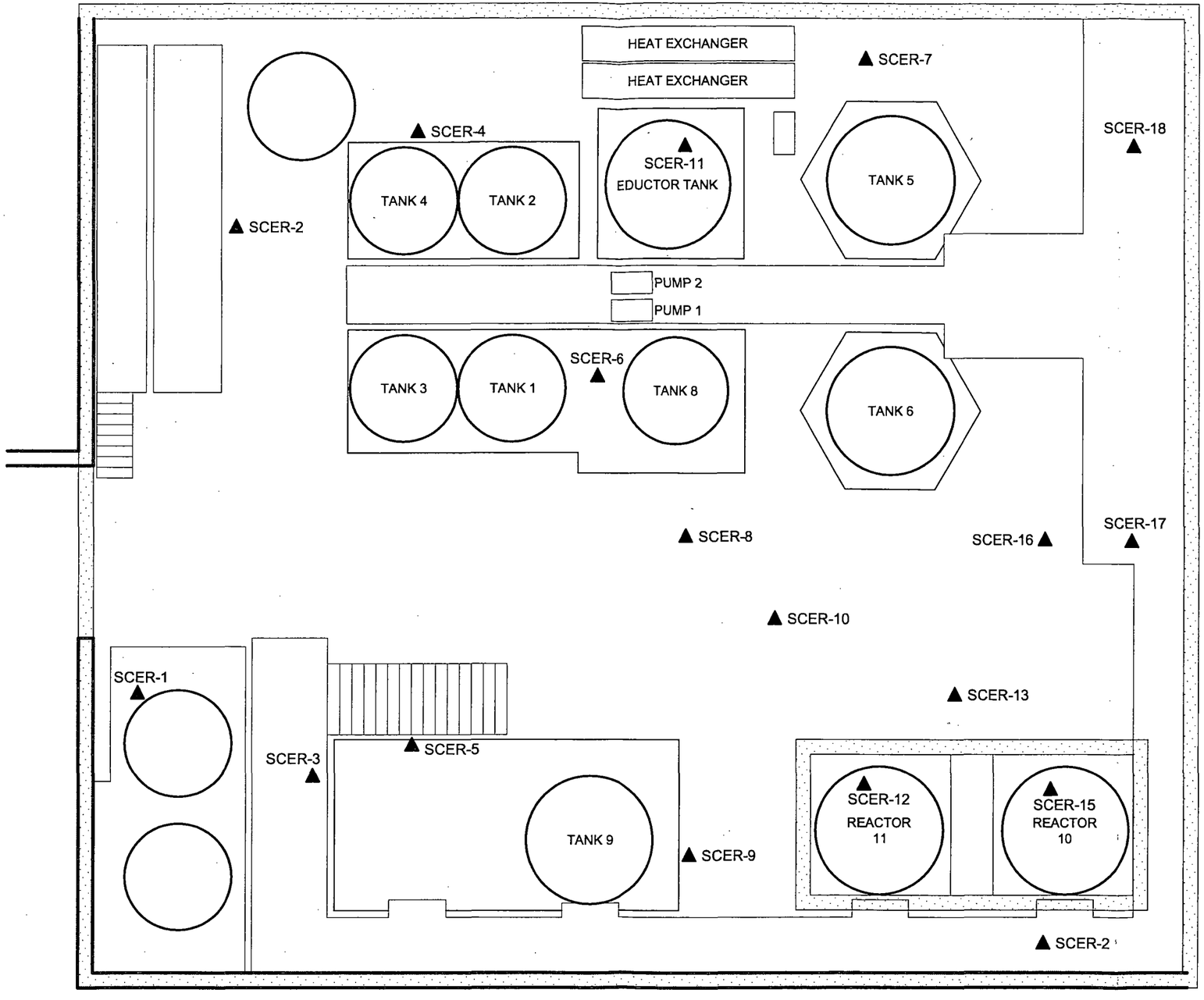
SOURCE: Map created with TOPO! ® ©2001 National Geographic (www.nationalgeographic.com/topo)

0 2000 4000

SCALE, FEET

MacDERMID, INC. 526 HUNTINGDON AVENUE WATERBURY, CONNECTICUT	GEI Consultants	**SITE LOCATION MAP**
MacDERMID, INC. WATERBURY, CONNECTICUT	Project 073290-1000	April 2008 Figure 1

GEI\MacDERMID\MacDermid Location Map.cdr



LEGEND:

▲ SCER-1 CONCRETE CHIP SAMPLE LOCATION

SOURCE:

DRAWING BASED ON *FIGURE 5: SPENT COPPER ETCHANT RECYCLING, AREA D, MacDERMID INC., 526 HUNTINGDON AVENUE, WATERBURY, CT.,* PREPARED BY LEA, DATE: 12/04/02, SCALE: 1" = 10'.

0 10 20

SCALE, FEET

RCRA CLOSURE (AREA D)
526 HUNTINGDON AVENUE
WATERBURY, CONNECTICUT

MacDERMID, INC.
WATERBURY, CONNECTICUT

GEI Consultants

Project 073290-1001

SAMPLE LOCATIONS (SCER-1 THROUGH SCER-18) AREA D

March 2009 Figure 2

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Appendix A

Ceiling Values from CTDEP Draft RSR Revisions August 11, 2008

(New) Sections 22a-133k-1 through 22a-133k-3 of the Regulations of Connecticut State Agencies are amended by adding Appendix N as follows:

Appendix N to
Sections 22a-133k-1 through 22a-133k-3 of the Regulations of Connecticut State Agencies
Ceiling Values

	Volatile	Semivolatile	Inorganics	Pesticides	Units
Direct Exposure Criteria: Residential	500	1000	50000	500	mg/kg
Direct Exposure Criteria: Industrial/ Commercial	1000	2500	50000	1000	mg/kg
Groundwater Protection Criteria	1000	1000	1000	1000	ug/L
Pollutant Mobility Criteria					
Surface Water Protection Criteria	10000	10000	10000	10000	ug/L
Target Indoor Air Concentrations	500				ug/m^3
Volatilization Criteria: Groundwater	50000				ug/L
Volatilization Criteria: Soil Vapor	500				ug/m^3

Appendix B

Chloroethane Calculations and IRIS Data

Additional Chloroethane- RSR Criteria Calculations

Input Parameters

Direct Exposure Criteria Parameters			
RISK	Target Cancer Risk Level	Unitless	0.000001
HI	Hazard Index	Unitless	1
RfC	Reference Inhalation Concentration (IRIS)	mg/m^3	10
RfD	Reference Dose (Calculated for RfC)	$(mg/kg\text{-}day)^{-1}$	2.857
IR	Ingestion Rate	mg/day	50
EF	Exposure Frequency	days/year	250
ED	Exposure Duration	years	25
CF	Conversion Factor	kg/mg	0.000001
BW	Body Weight	kg	70
AT	Averaging Time, for carcinogens	days	25550
AT_A	Averaging Time, Adult non-carcinogens	days	9125
Pollutant Mobility Parameters			
RfD	Reference Dose (from IRIS)	$(mg/kg\text{-}day)^{-1}$	2.857
HI	Hazard Index	Unitless	1
BW	Body Weight	kg	70
AT	Averaging Time	days	25550
SA	Source Allocation	Unitless	0.2
IR	Ingestion Rate	l/day	2
EF	Exposure Frequency	days/year	365
ED	Exposure Duration	years	70
CF	Conversion Factor	Unitless	0.001

Calculations

Soil Matrix	**Value**	**Units**
Industrial/commercial direct exposure criteria (I/C DEC)		
DEC_{RB} = (RFD *HI)* ((BW*AT)/(IR*EF*ED*CF))	16352000	mg/kg
Groundwater Pollutant Mobility Criteria for GB Areas (GB PMC)		
GB PMC = GWPC * 20 (µg/L to µg/kg) * 10 (Conversion from GA to GB)	4000000	µg/kg
Groundwater Matrix		
Groundwater Protection Criteria (GWPC)		
GWPC = (RFD x HI) x ((BW x AT x SA) / (IR x EF x ED x CF))	20000	µg/L

Notes:

Reference Dose collected from EPA IRIS website at www.epa.gov/iris/ Accessed March 11, 2009

GWPC and I/C DEC formulas referenced to the Nov. 18, 2002 memo for CTDEP Corrected Criteria Formula

http://www.epa.gov/NCEA/iris/subst/0523.htm
Last updated on Thursday, January 10th, 2008.

Integrated Risk Information System

Ethyl chloride (CASRN 75-00-3)

view QuickView

MAIN CONTENTS

Search IRIS by Keyword

List of IRIS Substances

IRIS Summaries/Toxicological Reviews

Entire IRIS Website

Reference Dose for Chronic Oral Exposure (RfD)

0523

Ethyl chloride; CASRN 75-00-3

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR Ethyl chloride

File First On-Line 04/01/1991

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	no data	
Inhalation RfC Assessment (I.B.)	on-line	04/01/1991
Carcinogenicity Assessment (II.)	no data	01/01/1995

I. Chronic Health Hazard Assessments for Noncarcinogenic Effects

I.A. Reference Dose for Chronic Oral Exposure (RfD)

Substance Name — Ethyl chloride
CASRN — 75-00-3
Primary Synonym — Chloroethane

Not available at this time.

_I.B. Reference Concentration for Chronic Inhalation Exposure (RfC)

Substance Name — Ethyl chloride
CASRN — 75-00-3
Primary Synonym — Chloroethane
Last Revised — 04/01/1991

The inhalation Reference Concentration (RfC) is analogous to the oral RfD and is likewise based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. The inhalation RfC considers toxic effects for both the respiratory system (portal-of-entry) and for effects peripheral to the respiratory system (extrarespiratory effects). It is expressed in units of mg/cu.m. In general, the RfC is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily inhalation exposure of the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Inhalation RfCs were derived according to the Interim Methods for Development of Inhalation Reference Doses (EPA/600/8-88/066F August 1989) and subsequently, according to Methods for Derivation of Inhalation Reference Concentrations and Application of Inhalation Dosimetry (EPA/600/8-90/066F October 1994). RfCs can also be derived for the noncarcinogenic health effects of substances that are carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

__I.B.1. Inhalation RfC Summary

Critical Effect	Exposures*	UF	MF	RfC
Delayed fetal ossification	NOAEL: 4000 mg/cu.m (1504 ppm) NOAEL(ADJ): 4000 mg/cu.m NOAEL(HEC): 4000 mg/cu.m	300	1	1E+1 mg/cu.m
Mouse Developmental Inhalation study Scortichini et al., 1986	LOAEL: 13,000 mg/cu.m (4946 ppm) LOAEL(ADJ): 13,000 mg/cu.m LOAEL(HEC): 13,000 mg/cu.m			

*Conversion Factors: MW = 64.5. Assuming 25C and 760 mm Hg, NOAEL (mg/cu.m) = 1504 ppm x MW/24.45 = 4000 mg/cu.m. For developmental effects this concentration is not adjusted; therefore NOAEL(ADJ) = NOAEL. The NOAEL (HEC) was calculated for a gas:extrarespiratory effect assuming periodicity was attained. b:a lambda(a) is unknown, b:a lambda(h) = 2.69, (Gargas et al., 1989). Since b:a lambda(a) is unknown, a default value of 1.0 is used for this ratio. NOAEL(HEC) = NOAEL(ADJ) x 1 = 4000 mg/cu.m.

__I.B.2. Principal and Supporting Studies (Inhalation RfC)

Scortichini, B.H., K.A. Johnson, J.J. Momany-Pfruender, and T.R. Hanley, Jr. 1986. Ethyl chloride: Inhalation teratology study in CF-1 mice. Dow Chemical Co. EPA Document #86-870002248.

In a developmental study conducted in groups of 30 CF-1 mice, Scortichini et al. (1986) exposed animals to mean time-weighted averages of 0 (air), 491 +/-37 ppm (1.3 g/cu.m), 1504 +/- 84 ppm (4000 mg/cu.m), and 4946 +/- 159 ppm (13,000 mg/cu.m.) 99.9% ethyl

chloride for 6 hours/day on days 6 through 15 of gestation. The animals were sacrificed on the 18th day of gestation. In accordance with current EPA practice these values are not duration adjusted. No maternal toxicity was recorded in this study (clinical signs, body weight, liver weight, and food and water consumption were monitored), although an earlier pilot study with non-pregnant female mice at these same concentrations showed an exposure-related decrease in body weight gain (data not presented). In the present study, no exposure-related changes were noted in resorption rate, litter size, sex ratios, or fetal body weights. No exposure-related fetal visceral malformations were observed. In the fetuses of the dams exposed to 4946 ppm, there was a statistically significant increased incidence ($p < 0.05$) of foramina of the skull bones, a small area of delayed ossification. At this concentration, 5 fetuses were affected in a total of 5 litters vs. 1 fetus in 1 litter in the controls and in each lower exposure group (the skull bones were examined in 22 to 25 litters in the controls and at each exposure level). The authors cite that the historical incidence of foramina of the skull bones in their facility with this strain of mice is 0.2% of the fetuses with a range of 0 to 1.2% The effect in this study at 4946 ppm ethyl chloride represented 4% of the fetuses. Additional information volunteered by one author (TRH) indicated that the foramina in question were small, pin-point lesions although apparently the openings were not measured. This skull effect was accompanied by an increasing incidence of cervical ribs (a supernumerary rib considered to be a malformation). The incidence of fetuses having this malformation was 2/257 (1%) of the controls, and, in order of increasing exposure concentrations, 1/299 (0.3%), 6/311 (2%), and 4/242 (2%). The corresponding figures for the incidence in litters was 2/22 (9%) in controls and 1/25 (4%), 5/26 (19%), and 4/22 (18%) in the litters of exposed dams. This effect was not indicated as statistically significant and no historical incidence for this malformation is given in the text. This study shows that exposure to ethyl chloride results in fetotoxicity. The exposure concentration of 1504 ppm is the NOAEL of this study NOAEL(HEC) = 4000 mg/cu.m based on foramina of the skull bones. The highest concentration used in this study, 4946 ppm, is a LOAEL, (HEC) = 13,000 mg/cu.m.

__I.B.3. Uncertainty and Modifying Factors (Inhalation RfC)

UF — A factor of 10 is used to account for sensitive populations. An uncertainty factor of 3 (rather than 10) is used for interspecies extrapolation due to dosimetric adjustment of the inhaled concentration. As no multigeneration reproductive study and no definitive developmental toxicity studies were available, a full factor of 10 is proposed for database deficiencies.

MF — None

__I.B.4. Additional Studies/Comments (Inhalation RfC)

Although used as a surgical anesthetic, ethyl chloride has a narrow margin of safety for this purpose as anesthesia occurs at 20 to 30 mg% and respiratory failure at 40 mg% (Dobkin and Byles, 1971). Ethyl chloride is explosive at 4% (40,000 ppm, 106 g/cu.m) in air, overlapping the concentrations required to produce anesthesia (3 to 4.5%). Neurological symptoms have been observed in human case-studies in instances of ethyl chloride abuse. Hes et al. (1979) noted cerebellar-related symptoms including ataxia, tremors, dysarthria (speech difficulties), slowed reflexes, nystagmus (involuntary movement of the eyeball), and hallucinations in a 28-year old female who sniffed 200 to 300 mL of ethyl chloride off her coat sleeve daily for 4 months. Examination revealed that her liver was enlarged (3 cm) and slightly tender and was accompanied by a mild and transient disturbance (not clinically described) of liver function. All symptoms were resolved by the end of 4 weeks. Similar neurological symptoms were noted in a 52-year old male who had a 30-year history of

intermittent ethyl chloride (as well as alcohol and barbiturate) abuse (Nordin et al., 1988). Questioning upon hospitalization revealed that he had been inhaling at least 100 mL of ethyl chloride daily for the previous 4 months. No liver effects were reported and the patient fully recovered from the neurological symptoms by 6 weeks after admission. Ethyl chloride has been demonstrated to be a cardiac sensitizer (Balazs, et al., 1986) in dogs at or near concentrations producing anesthesia, i.e., 30,000 to 45,000 ppm (du Pont, 1971). In this condition, cardiac tissue is hypersensitized to the effects of stimulatory endogenous catecholamines which can result in arrhythmias and cardiac arrest.

Rowe et al. (1939) exposed groups of rabbits (4/group) and rats (12/group; strain unspecified) to 26.4 g/cu.m ethyl chloride 7.5-8 hours/day, 5 days/week for 6.5 months. No effects on weight gain, liver weights, histopathology (including lungs), or clinical signs were noted.

Landry et al. (1989) exposed groups of 14 (7/sex) B6C3F1 mice to 0 (air), 250 ppm (0.66 g/cu.m), 1247 ppm (3.3 g/cu.m), or 4843 ppm (12.8 g/cu.m) 99.9% EC, 23 hours/day for 11 consecutive days. The duration-adjusted values for these exposures in increasing concentrations are 0, 0.63, 3.2, and 12.2 g/cu.m. The actual duration of exposure in this study (253 hours) was comparable to that obtained in a 4 hour/day, 5-day exposure week (260 hours). A blind neurobehavioral observation battery was conducted on the 12th day followed by collection of samples for clinical chemistry and hematology. Body and organ weights were taken and histopathology was performed. The only exposure-related effect observed in this study was a slight increase in the mean liver weights of both male and female mice exposed to 4843 ppm. (The increase in liver weight was approximately 6 g/100g vs. 5.3 g/100 g in controls; p=0.05.) Histopathologic examination revealed a minimal increase in the degree of hepatocellular vacuolization in 4 of 7 animals of both sexes at this exposure. These alterations were minimal and not accompanied by any increase in serum enzymes. This study defines a free-standing NOAEL of 4843 ppm, the NOAEL(HEC) for this extrarespiratory effect = 12.2 g/cu.m.

Landry et al. (1982) exposed groups of 8-10 week old F344 rats (6/sex/group) to 0 (air), 1600 ppm (4.2 g/cu.m), 4000 ppm (10.6 g/cu.m), or 10000 ppm (26.4 g/cu.m) of 99.7% ethyl chloride 6 hours/day, 5 days/week for 2 weeks. The duration-adjusted values are 0, 0.8, 1.9, or 4.7 g/cu.m, respectively. Clinical observations and chemistry, hematology, urinalysis, and complete histopathology (including the entire respiratory tract) were performed. The only exposure-related effect observed was a statistically significant increase in liver to body weight ratios in male rats exposed to 4000 ppm (3.64 g/100g) and 10,000 ppm (3.73 g/100g) as compared with controls (3.47 g/100 g) ethyl chloride. As this alteration was not accompanied by any histopathology or increases in serum enzymes it is considered an adaptive response, not an adverse effect. Therefore this study identifies the highest level of exposure in this study (10,000 ppm) as a free-standing NOEL, NOEL(HEC) for extrarespiratory effects = 4.7 g/cu.m.

Groups of F344 rats and B6C3F1 mice (50/group/sex) were exposed to either 0 (air) or 15,000 ppm of 99.5% ethyl chloride (39.6 g/cu.m) 5 days/week, 6 hours/day for 102 weeks (rats) or 100 weeks (mice) in an NTP (1989) study. The duration-adjusted concentration becomes 7.1 g/cu.m. The exposure level was set at this limit because of safety considerations for explosions. A single level of exposure was chosen as no exposure-related changes were seen in the 90-day study (see below) at a slightly higher concentration (19,000 ppm). Monitoring for toxicological effects was by twice daily observation, body weights, and a complete necropsy and histologic examination including tissues of the entire respiratory tract (3 levels of the nasal epithelium, personal communication with study director) and brain. Survival of female mice after week 82 was significantly lower than

controls apparently due to an increase in deaths from carcinomas of the uterus; there were no other statistically significant differences in survival between control and treated animals of either species. The incidences and severity of microscopic pathologies noted in tissues (including uterine tissue) were not different between the treated and control animals of either species. Hyperactivity was observed but only in female mice (no incidences given) and only during exposure. Mean body weights were decreased in both male and female rats. In females, the maximum difference in body weights between exposed and control animals was 13% and occurred at 59 weeks of exposure when 49 of 50 test animals were still alive. Although some fluctuations towards normalcy were observed from this time forward, terminal body weights of 23 surviving treated animals were still 10% less than their corresponding controls. In male rats, mean body weights were also decreased when compared with controls, although the decrease achieved a maximum differential of only 8%. The mean body weights of mice were not affected by exposure. Based on the mild decrease in mean body weight gain, 15,000 ppm is judged as a free-standing NOAEL. The NOAEL(HEC) = 7.1 g/cu.m.

Groups of F344 rats and B6C3F1 mice (10/group) were exposed to either 0 (air), 2500 ppm (6.6 g/cu.m), 5000 ppm (13.2 g/cu.m), 10,000 ppm (26.4 g/cu.m), or 19,000 ppm (50.1 g/cu.m) of 99.5% ethyl chloride 5 days/week, 6 hours/day for 13 weeks (NTP, 1989). The duration-adjusted concentrations are 0, 1.2, 2.4, 4.7, or 9.0 g/cu.m, respectively. Monitoring for toxicological effects was by daily observation, body weights, and a complete necropsy and histologic examination including tissues of the entire respiratory tract and brain. No exposure-related clinical signs or gross or histopathological effects were observed in either species. Relative liver weights were slightly increased in the male rats (14%) and female mice (18%) exposed to 19,000 ppm. Slight decreases in mean body weights were noted in the rats (8% in the males, 4% in the females) exposed to 19,000 ppm; no dose-related tendency could be discerned from the data. As no toxicity was apparent, 19,000 ppm is considered as a free-standing NOAEL in this study. The NOAEL(HEC) = 9.0 g/cu.m.

The results obtained in the two studies of Troshina (1964 & 1966) discussed below do not concur with those found by NTP (1989), Landry et al. (1982, 1989), or Rowe et al. (1939). All of the latter are carefully conducted studies with appropriate controls and relatively complete presentation and description of the data obtained. As presented, the studies of Troshina may be described as ambiguously conducted with deficient use of controls and no or little presentation of data. These deficiencies preclude consideration of these studies as a reliable source of information about the toxic effects of this chemical.

In the study published in 1964, Troshina exposed 12 rats (sex or strain not specified) for 2 hours/day for 60 days (assumed consecutive) 14 g/cu.m ethyl chloride, the duration-adjusted value being 1.2 g/cu.m. There is mention of but no description of controls used in this study. Body weight, hematology, some histopathology, and the "functional state of the nervous system and the liver" were assessed for adverse effects. Body weights were unaffected. Using a functional test of liver metabolic capacity (conversion of gastrically administered sodium benzoate to hippuric acid as measured by urinary excretion), a decrease in hippuric acid excretion was noted after the exposure, from 90.3% in controls to 33.6% in the exposed animals. Lung pathology was described as bronchitis, hyperemia, and (apparently) intraalveolar thickening. The author claims these effects are exposure-related indications of irritant action, although no mention is made of histology from control lungs. Description of liver pathology included nodule formation originating from the reticuloendothelial cells while "very slight" adiposity was also noted. Belying this description of substantial pathology, the author states that these changes were "weakly pronounced." After noting a increased tendency of exposed animals to form cutaneous abscesses (4 of 12), the authors examined other animals (apparently exposed under identical conditions for 2

weeks, n = at least 3) for decrements in phagocytic activity. Their data showed a decrease in phagocyte number, index (not described), and percent of active cells at the end of the 2-week period, although evaluation past this early time point was apparently not done. No scientific conclusions could be reliably drawn from this study, although effects would suggest the exposure level of 1.2 g/cu.m to be a frank-effect level (FEL). For extrarespiratory effects, FEL(HEC) = 1.2 g/cu.m. The FEL(HEC) was also calculated for a gas:respiratory effect in the thoracic region. MVa = 0.14 cu.m/day, MVh = 20 cu.m/day, Sa(TH) = 3461.6 sq.cm., Sh(TH) = 640581 sq.cm. RGDR = (MVa/Sa) / (MVh/Sh) = 1.3. FEL(HEC) = FEL(ADJ) x RGDR = 1.6 g/cu.m.

In the 1966 study by the Troshina, exposures were lowered substantially from the 1964 experiments (presumably due to the frank effects) and are reported as 0, 0.06, or 0.57 g/cu.m in exposures to 12 rats which lasted for 6 months at 4 hours/day, 6 days/week. The duration-adjusted values would be 0, 0.0085, or 0.0811 g/cu.m. Using the same indicators of toxicity as in the 1964 study, the author reported decreases in phagocytic activity although these indices "fluctuated within considerable limits." Although no data are presented, the author also describes several exposure-related effects including disturbed liver function, lowered blood pressure, fatty liver, and what is interpreted as intraalveolar thickening in the lungs. No scientific conclusions could be reliably drawn from this study, although effects claimed would suggest the exposure level of 0.0085 g/cu.m = 8.5 mg/cu.m = NOAEL(HEC) based on extrarespiratory effects. The NOAEL(HEC) was also calculated for a gas:respiratory effect in the pulmonary region. MVa = 0.14 cu.m/day, MVh = 20 cu.m/day, Sa(PU) = 3424 sq.cm., Sh(TH) = 635545 sq.cm. RGDR = (MVa/Sa) / (MVh/Sh) = 1.3. NOAEL(HEC) = NOAEL(ADJ) x RGDR = 11.1 mg/cu.m.

Experiments conducted by Breslin et al. (1988) suggest that exposure to ethyl chloride may disrupt the estrus cycle of mice. Two groups (10/group) of female B6C3F1 mice were acclimated in exposure chambers over a 2-week period or until the estrus cycles of most mice was a 4-6 day interval (as judged by a vaginal lavage technique). Males were included in each chamber to synchronize and promote regular estrus cyclicity. Following acclimatization one group was exposed to 15,000 ppm (39.6 g/cu.m) ethyl chloride 6 hours/day for a minimum of 14 consecutive days (through 3 estrus cycles). No effects on behavior, gross or histopathology were observed in the group undergoing exposure although the mean body weights in the exposed group was significantly increased rather than decreased. The mean length of the estrus cycle in exposed mice was 5.6 days, significantly longer in duration than the pre- exposure duration for the same group (5.0 days) and for the corresponding controls (4.5 days). The protraction of the period could not be attributed to an increase in any particular phase of the estrus cycle and is therefore suggestive of a general stress response. A direct exposure-related effect of ethyl chloride on neuroendocrine function cannot be excluded. As this effect is regarded as a systemic effect, the exposure is duration adjusted to establish a free-standing LOAEL of 6.6 g/cu.m. The LOAEL(HEC) = 6.6 g/cu.m.

I.B.5. Confidence in the Inhalation RfC

Study — Medium
Database — Medium
RfC — Medium

Although the principal study is well-conducted, it does not establish a firm concentration-response relationship with an adverse effect and was not performed at levels eliciting maternal toxicity. There are no multigenerational reproductive studies for this compound, and without a developmental study in a second species, the overall confidence in the data base is medium. Medium confidence in the RfC follows.

__I.B.6. EPA Documentation and Review of the Inhalation RfC

Source Document — This assessment is not presented in any existing U.S. EPA document.

Other EPA Documentation — U.S. EPA, 1987, 1988

Agency Work Group Review — 12/20/1990

Verification Date — 12/20/1990

__I.B.7. EPA Contacts (Inhalation RfC)

Please contact the IRIS Hotline for all questions concerning this assessment or IRIS, in general, at (202)566-1676 (phone), (202)566-1749 (FAX) or hotline.iris@epa.gov (internet address).

_II. Carcinogenicity Assessment for Lifetime Exposure

Substance Name — Ethyl chloride
CASRN — 75-00-3
Primary Synonym — Chloroethane

Not available at this time.

_VI. Bibliography

Substance Name — Ethyl chloride
CASRN — 75-00-3
Primary Synonym — Chloroethane
Last Revised — 04/01/1991

_VI.A. Oral RfD References

None

_VI.B. Inhalation RfC References

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_VI.C. Carcinogenicity Assessment References

None

_VII. Revision History

Substance Name — Ethyl chloride
CASRN — 75-00-3
Primary Synonym — Chloroethane

Date	Section	Description
04/01/1991	I.B.	Inhalation RfC summary on-line
04/01/1991	VI.	Bibliography on-line
01/01/1992	IV.	Regulatory Action section on-line
01/01/1995	II.	Carcinogenicity assessment now under review
08/01/1995	II.	EPA's RfD/RfC and CRAVE workgroups were discontinued in May, 1995. Chemical substance reviews that were not completed by September 1995 were taken out of IRIS review. The IRIS Pilot Program replaced the workgroup functions beginning in September, 1995.
04/01/1997	III., IV., V.	Drinking Water Health Advisories, EPA Regulatory Actions, and Supplementary Data were removed from IRIS on or before April 1997. IRIS users were directed to the appropriate EPA Program Offices for this information.
01/02/1998	I., II.	This chemical is being reassessed under the IRIS Program.

_VIII. Synonyms

Substance Name — Ethyl chloride
CASRN — 75-00-3
Primary Synonym — Chloroethane
Last Revised — 07/01/1995

- 75-00-3
- Ethane, chloro-
- Aethylchlorid [German]
- Aethylis
- AETHYLIS CHLORIDUM
- Anodynon
- Chelen
- Chloorethaan [Dutch]
- Chlorene
- Chlorethyl

- Chloridum
- Chloroaethan [German]
- Chloroethane
- Chlorure d'ethyle [French]
- Chloryl
- CHLORYL ANESTHETIC
- Cloretilo
- Cloroetano [Italian]
- Cloruro de etilo [Spanish]
- CLORURO DI ETILE [Italian]
- Dublofix
- ETHANE, CHLORO-
- ETHER CHLORATUS
- ETHER HYDROCHLORIC
- ETHER MURIATIC
- Ethyl Chloride
- ETYLU CHLOREK [Polish]
- HSDB 533
- Hydrochloric ether
- Kelene
- Monochlorethane
- Monochloroethane
- Muriatic ether
- Narcotile
- NCI-CO6224
- UN 1037

IRIS Home

Chronic Health Hazards for Non-Carcinogenic Effects

Reference Dose for Chronic Oral Exposure (RfD)

- Oral RfD Summary
- Principal and Supporting Studies
- Uncertainty and Modifying Factors
- Additional Studies/Comments
- Confidence in the Oral RfD
- EPA Documentation and Review

Reference Concentration for Chronic Inhalation Exposure (RfC)

- Inhalation RfC Summary
- Principal and Supporting Studies
- Uncertainty and Modifying Factors

Appendix C

2-Hexanone Calculations

Additional Hexanone,2-RSR Criteria Calculations

Input Parameters

Direct Exposure Criteria Parameters			
RISK	Target Cancer Risk Level	Unitless	0.000001
HI	Hazard Index	Unitless	1
RFD	Reference Dose (1993 superfund Technical Support Center)	$(mg/kg\text{-}day)^{-1}$	0.040
IR	Ingestion Rate	mg/day	50
EF	Exposure Frequency	days/year	250
ED	Exposure Duration	years	25
CF	Conversion Factor	kg/mg	0.000001
BW	Body Weight	kg	70
AT	Averaging Time, for carcinogens	days	25550
AT_A	Averaging Time, Adult non-carcinogens	days	9125
Pollutant Mobility Parameters			
RFD	Reference Dose	$(mg/kg\text{-}day)^{-1}$	0.040
HI	Hazard Index	Unitless	1
BW	Body Weight	kg	70
AT	Averaging Time	days	25550
SA	Source Allocation	Unitless	0.2
IR	Ingestion Rate	l/day	2
EF	Exposure Frequency	days/year	365
ED	Exposure Duration	years	70
CF	Conversion Factor	Unitless	0.001

Calculations

Soil Matrix	**Value**	**Units**
Industrial/commercial direct exposure criteria (I/C DEC)		
DEC_{RB} = (RFD *HI)* ((BW*AT)/(IR*EF*ED*CF))	228928	mg/kg
Groundwater Pollutant Mobility Criteria for GB Areas (GB PMC)		
GB PMC = GWPC * 20 (µg/L to µg/kg) * 10 (Conversion from GA to GB)	56000	µg/kg
Groundwater Matrix		
Groundwater Protection Criteria (GWPC)		
GWPC = (RFD x HI) x ((BW x AT x SA) / (IR x EF x ED x CF))	280	µg/L

Notes:

GWPC and I/C DEC formulas referenced to the Nov. 18, 2002 memo for CTDEP Corrected Criteria Formula

Appendix D

Methyl Methacrylate Calculations and IRIS Data

Additional Methyl methacrylate RSR Criteria Calculations

Input Parameters

Direct Exposure Criteria Parameters			
RISK	Target Cancer Risk Level	Unitless	0.000001
HI	Hazard Index	Unitless	1
RFD	Reference Dose (From IRIS)	$(mg/kg\text{-}day)^{-1}$	1.400
IR	Ingestion Rate	mg/day	50
EF	Exposure Frequency	days/year	250
ED	Exposure Duration	years	25
CF	Conversion Factor	kg/mg	0.000001
BW	Body Weight	kg	70
AT	Averaging Time, for carcinogens	days	25550
AT_A	Averaging Time, Adult non-carcinogens	days	9125
Pollutant Mobility Parameters			
RFD	Reference Dose (from IRIS)	$(mg/kg\text{-}day)^{-1}$	1.400
HI	Hazard Index	Unitless	1
BW	Body Weight	kg	70
AT	Averaging Time	days	25550
SA	Source Allocation	Unitless	0.2
IR	Ingestion Rate	l/day	2
EF	Exposure Frequency	days/year	365
ED	Exposure Duration	years	70
CF	Conversion Factor	Unitless	0.001

Calculations

Soil Matrix	**Value**	**Units**
Industrial/commercial direct exposure criteria (I/C DEC)		
DEC_{RB} = (RFD *HI)* ((BW*AT)/(IR*EF*ED*CF))	8012480	mg/kg
Groundwater Pollutant Mobility Criteria for GB Areas (GB PMC)		
GB PMC = GWPC * 20 (µg/L to µg/kg) * 10 (Conversion from GA to GB)	1960000	µg/kg
Groundwater Matrix		
Groundwater Protection Criteria (GWPC)		
GWPC = (RFD x HI) x ((BW x AT x SA) / (IR x EF x ED x CF))	9800	µg/L

Notes:

Reference Dose collected from EPA IRIS website at www.epa.gov/iris/ Accessed March 11, 2009

GWPC and I/C DEC formulas referenced to the Nov. 18, 2002 memo for CTDEP Corrected Criteria Formula

http://www.epa.gov/ncea/iris/subst/1000.htm
Last updated on Tuesday, January 15th, 2008.

Integrated Risk Information System

Methyl methacrylate (CASRN 80-62-6)

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Reference Dose for Chronic Oral Exposure (RfD)

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Note: A TOXICOLOGICAL REVIEW is available for this chemical in Adobe PDF Format (83 Pages, 275 Kbytes). Similar documents can be found in the List of Available IRIS Toxicological Reviews.

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Methyl methacrylate; CASRN 80-62-6 (03/02/98)

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR Methyl methacrylate

File First On-Line 03/02/98

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	on-line	03/02/98*
Inhalation RfC Assessment (I.B.)	on-line	03/02/98*
Carcinogenicity Assessment (II.)	on-line	03/02/98*

*A comprehensive review of toxicological studies was completed (June 5, 2006) - please see section I.A.6., I.B.6., II.D.2. for more information.

_I. Chronic Health Hazard Assessments for Noncarcinogenic Effects

_I.A. Reference Dose for Chronic Oral Exposure (RfD)

Substance Name — Methyl methacrylate
CASRN — 80-62-6
Last Revised — 03/02/98

The oral Reference Dose (RfD) is based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. It is expressed in units of mg/kg-day. In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Please refer to the Background Document for an elaboration of these concepts. RfDs can also be derived for the noncarcinogenic health effects of substances that are also carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

__I.A.1. Oral RfD Summary

Critical Effect	Experimental Doses	UF	MF	RfD
None Rat drinking water study Borzelleca et al. (1964)	NOAEL:136 mg/kg/day	100	1	1.4 mg/kg/day

__I.A.2. Principal and Supporting Studies (Oral RfD)

Borzelleca, JF; Larson, PS; Hennigar, GR, Jr; Huf, EG; Crawford, EM; Smith, RB, Jr., (1964) Studies on the chronic oral toxicity of monomeric ethyl acrylate and methyl methacrylate. Toxicol. Appl. Pharmacol. 6:29-36.

Borzelleca et al. (1964) exposed groups of 25 male and 25 female Wistar rats to MMA in drinking water continuously for 104 weeks. The initial exposure concentrations were 6, 60, and 2,000 ppm MMA. The low and medium exposures were increased to 7 and 70 ppm, respectively, at the start of the fifth month, resulting in TWA exposure concentrations of 6.85 and 68.46 ppm MMA. Survival of exposed rats was not significantly different from controls. An initial reduction in body weight gain was observed in both males and females exposed to 2,000 ppm MMA; this reverted to control levels by week 3 (females) and week 6 (males). This is likely the result of reported reduced food intake during the first month, which was not observed in the second month and beyond. No other effects on body weight gain were reported, but drinking water consumption was significantly lower than controls in males and particularly females of the high-exposure groups. Hematological parameters were normal throughout the study in all groups, and no compound-related effects were observed on urinary protein or reducing substances. No abnormalities or lesions related to MMA were identified from histopathological examination of the tissues of exposed rats. The only effect observed was an increased kidney/body-weight ratio in female rats exposed to 2,000 ppm MMA, but the increase was only marginally significant and was not associated with any histopathological findings. Thus, the highest exposure level, 136 mg/kg/day (2,000 mg/L × 0.0313 L/rat/day divided by

the default body weight for Wistar rats of 0.462 kg), is considered a NOAEL for this study.

__I.A.3. Uncertainty and Modifying Factors (Oral RfD)

UF — 100.

The following uncertainty factors are applied to this effect level: 10 for consideration of intraspecies variation (UF_H; human variability), a partial uncertainty factor of 3 for extrapolation for interspecies differences (UF_A; animal to human), and an uncertainty factor of 3 to account for a deficient database (UF_D). The total UF = 10 × 3 × 3 = 100.

A full uncertainty factor for intraspecies differences (UF_H) was used to account for potentially sensitive human subpopulations. This UF was not reduced because of the lack of human oral exposure information.

A partial threefold uncertainty factor to account for laboratory animal-to-human interspecies differences (UF_A) was used. The slower blood metabolism of MMA in humans (Bereznowski, 1995), combined with the fact that humans do not have a forestomach (target organ in the Borzelleca et al., 1964 study) lowers the potential for a more pronounced portal-of-entry effect in humans. However, complete elimination of this UF is not justified, given the lack of human oral exposure information and remaining uncertainty regarding MMA's potential to cause other effects in humans following chronic oral exposure.

The major areas of uncertainty in this assessment are the lack of an identified critical effect to humans, the lack of a chronic study in a second species, the lack of a neurologic study, and the lack of a developmental or reproductive toxicity study via the oral route (given that developmental effects have been seen in laboratory animals following other routes of exposure). A partial three-fold database uncertainty factor (UF_D) was employed, however, because a number of repeat exposure inhalation studies, including developmental, reproductive, and chronic studies, lend support to the oral database.

MF — 1.

__I.A.4. Additional Studies/Comments (Oral RfD)

There are three repeat exposure studies that were of long enough duration to be considered for use in the derivation of an oral RfD: the Motoc et al. (1971) rat study, the Borzelleca et al. (1964) rat study, and the Borzelleca et al. (1964) dog study. Of the three, only the Borzolleca et al. (1964) drinking water study in rats was of chronic duration (2 years). Motoc et al. (1971) was a subchronic gavage study, and the assessment of dogs by Borzelleca et al. (1964) involved the administration of MMA in gelatin capsules. The Motoc et al. (1971) gavage study showed that large bolus doses can overwhelm detoxification mechanisms and cause stomach ulcerations in rats. Thus, the less-than-chronic gavage studies of Motoc et al. (1971) and Borzelleca et al. (1964) were considered less desirable for use in the derivation of an RfD than the chronic drinking water study in rats of Borzelleca et al. (1964). Borzelleca et al. (1964) reported an increase in kidney-to-body ratios for female rats, but it was only marginally significant and was not associated with any histopathological findings. The fact that MMA was not reported to cause gastric toxicity in this study is not in and of itself a reason to doubt the results of the study. Substitution on the number 2 carbon of acrylic acid has been shown in gavage studies to abolish gastric toxicity (Ghanayem et al., 1985) and cell proliferation (Ghanayem et al., 1986).

Borzelleca et al. (1964) found no significant toxic effects in male and female dogs (2 males and 2 females per treatment group) receiving MMA via gelatin capsule in the diet at 10, 100, or

1,473 ppm daily for 1 year. The high exposure concentration represented a time-weighted average based on the 1,000 ppm value, increasing to 1,200 ppm at 5 weeks, to 1,400 ppm at 7 weeks, and to 1,500 ppm at 9 weeks.

Motoc et al. (1971) orally administered methyl methacrylate to albino rats for 3 (20 exposures), 5 (41 exposures), or 8 (63 exposures) months. Total doses were reported as 2,750, 5,500, and 8,125 mg/kg, respectively, for these exposure periods. The authors reported duration-related increases in histopathological alterations of the liver, ulcerations of the stomach, and biochemical alterations (elevated serum enzyme activity), but no further details were described.

The LD_{50} for MMA was estimated to be 8.41-10 mL/kg (7.87-9.36 g/kg) in rats, 6.3 mL/kg (5.9 g/kg) in guinea pigs, and 5 (4.68 g/kg) in dogs (Deichmann, 1941; Spealman et al., 1945). The lowest lethal concentration in rabbits administered MMA by gavage was 6.55 g/kg body weight. Toxic symptoms in both species included increased respiratory rate and motor weakness. These were followed by decreased respiration at 15 to 40 minutes post-administration, shallow and irregular respiration, increased urination and defecation, hemoglobinuria, loss of reflex activity, coma, and death. Adverse intestinal changes were observed in orally exposed animals.

Central nervous system effects were observed in Wistar rats given 500 mg/kg body weight/day MMA in olive oil by gavage for 21 days (Husain et al., 1985; Husain et al., 1989). Treated rats were observed to be lethargic and had gait defects and hind limb weakness for about 10 min after each treatment. Locomotor activity and learning ability were significantly decreased and aggressive behavior was significantly increased in exposed rats compared to controls.

No oral studies have investigated the developmental or reproductive toxicity of MMA. Evidence for developmental effects from inhalation exposure is mixed and generally occurred at maternally toxic exposure levels. Solomon et al. (1993) found no developmental effects in rats exposed 6 h/day during days 6-15 of gestation to atmospheric concentrations of up to 2,028 ppm (8,304 mg/m^3). Tansy (1979) and McLaughlin et al. (1978) found no developmental effects in mice exposed 6 h/day to up to 400 ppm and 2 h/day to 1,330 ppm, respectively, during days 6-15 of gestation. However, Nicholas et al. (1979) found evidence of developmental effects (early fetal deaths, delayed ossification, decreased fetal body weight and crown-rump length, hematomas) in Sprague-Dawley rats exposed for approximately 1 h/day during days 6-15 of gestation to levels more than an order of magnitude higher (110,000 mg/m^3). Nearly 20% of the exposed pregnant rats died at this exposure level. In addition, ICI (1977) and Luo et al. (1986) describe both delayed ossification and increased resorptions in rats exposed during days 6-15 of gestation to 1,000 ppm MMA (5 h/day and 2 h/3 days, respectively).

No adequate one- or two-generation reproductive studies were available by any route of exposure. MMA did not reveal an effect on male fertility in mice inhaling up to 9,000 ppm MMA for 6 h/day over a period of 5 days.

MMA is readily absorbed through the lungs, gastrointestinal tract, and skin. The experiments of Bratt and Hathway (1977) show that MMA is rapidly absorbed from the gastrointestinal tract of rats. Adult male Wistar rats were treated with 5.7 mg/kg ^{14}C-MMA by gavage. Up to 65% of the dose was expired from the lungs in 2 h, which shows the rapidity of the absorption. Recovery of radiolabel in the urine and feces accounted for only 7.4% of the administered dose, thereby indicating nearly complete absorption from the gastrointestinal tract. In addition, significant levels of methacrylic acid (> 0.5mM), a product of MMA degradation, were found in rat serum 5 min after a single dose of 8 mmol MMA/kg body weight (Bereznowski, 1995).

The only studies that provide definitive information regarding the distribution of MMA in a mammalian system following inhalation, oral, or intravenous exposures are those of Raje et al. (1985), Bratt and Hathway (1977), and Wenzel et al. (1973). Once absorbed, MMA is largely metabolized to methacrylic acid and eventually to CO_2 via the TCA cycle. In the experiments of Bratt and Hathway (1977), it was found that 10 days after oral or i.v. dosing of rats with ^{14}C-MMA, only 4.1%-6.6% ^{14}C-MMA remained in the carcass. That which is not metabolized to CO_2 and exhaled or excreted in the urine or feces is primarily retained in the liver and adipose tissue, though Raje et al. (1985) report finding small amounts of MMA in the brain and lungs following acute exposures.

Metabolism of MMA has been studied in vitro (Corkill et al., 1976; Bereznowski, 1995) and oral in vivo (Bratt and Hathway, 1977; Crout et al., 1982) in both rodents and humans. Several studies have confirmed the initial hydrolysis of MMA to methacrylic acid and methanol, and one in vitro study (Bereznowski, 1995) indicates that the rate of hydrolysis is slower in human than in rat blood. Available evidence suggests that MMA is enzymatically converted to methacrylic acid and is esterified to CoA, which is hydroxylated to -hydroxyisobutyric acid, oxidized and esterified by CoA to methylmalonyl CoA, and enters the citric acid cycle as succinyl CoA. Methacrylic acid, methyl malonic acid, ethyl malonic acid, b-hydroxyisobutyric acid, and mercapturic acid have been identified as urinary metabolites of the rat (Bratt and Hathway, 1977; Crout et al., 1982), and methyl malonic acid has been shown to be a urinary metabolite of humans (Crout et al., 1982).

Most of an orally or parenterally administered dose of ^{14}C-labeled MMA is excreted as CO_2 (Bratt and Hathway, 1977; Crout et al., 1982). Wistar rats given MMA orally, intraperitoneally, or intravenously exhaled 65%-86% of the administered radiolabel as CO_2 within 10 h of dosing. After 10 days, 88% and 84% of 5.7 mg/kg doses given orally and intravenously, respectively, were excreted as $^{14}CO_2$. An estimated 0.19%-1.4% of the administered dose was excreted by the lungs as unmetabolized MMA. The percent excreted as CO_2 decreased and the percent exhaled as unchanged MMA increased with increasing dose regardless of route (Bratt and Hathway, 1977). Urinary excretion accounted for about 4.7%-14.5% of the administered radioactivity (Bratt and Hathway, 1977; Crout et al., 1982), with about 0.22% of the radioactivity in the methylmalonic acid fraction (Crout et al., 1982). Other metabolites detected in the urine following oral or intravenous dosing with radiolabeled MMA include methacrylic acid, succinic acid, methylmalonic semialdehyde, -hydroxyisobutyric acid, and an unidentified ^{14}C-labeled acid. An estimated 1.7%-3% was excreted in feces following intragastric or intravenous administration (Bratt and Hathway, 1977). Methylmalonic acid was also detected in the urine of a human volunteer administered an ^{2}H-labeled dose of the sodium salt of MMA. ^{2}H-labeled methylmalonic acid was detected in the urine in an amount equal to about 1% of the administered dose (Crout et al., 1982).

For more detail on other Hazard Identification Issues, exit to the toxicological review, Section 4.7 (PDF)

I.A.5. Confidence in the Oral RfD

Study — Low to medium
Database — Low to medium
RfD — Low to medium

The overall confidence in the RfD assessment is low to medium. The confidence in the principal study is low to medium. The Borzelleca (1964) study is well documented, but does not appear to be conducted in accordance with what would now be considered Good Laboratory Practice and did not identify a LOAEL. Confidence in the database is judged to be low to medium.

Relevant, quantitative human subchronic or chronic studies are not available. Although repeat exposure inhalation studies, including developmental, reproductive, and chronic studies, bolster the weak and dated oral database somewhat, no developmental or reproductive studies are available by the oral route, and no multigenerational studies are available by any route of exposure. Gastrointestinal irritation has been identified in a rat subchronic gavage study (Motoc et al., 1971), but acute exposures to humans via the oral route are rare. Irritation is still considered the most likely effect of concern from oral exposure to humans, however, primarily because of extensive evidence from occupational studies and case reports that MMA is a respiratory irritant in humans.

For more detail on Characterization of Hazard and Dose Response, exit to the toxicological review, Section 6 (PDF).

__I.A.6. EPA Documentation and Review of the Oral RfD

Source Document — This assessment is presented in the Toxicological Review of Methyl Methacrylate. (CAS No. 80-62-6). (EPA, 1998)

U. S. Environmental Protection Agency. (1985) Health and environmental effects profile for methyl methacrylate. Cincinnati, OH: Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office; report no. EPA/600/X-85/364. Available from: NTIS, Springfield, VA; PB88-17885/XAB.

U.S. Environmental Protection Agency. (1988) Health and environmental effects profile for methyl methacrylate. NTIS/PB88-178785.

U.S. Environmental Protection Agency. (1991) Summary review of health effects associated with methyl methacrylate: health issue assessment. Environmental Criteria and Assessment Office, Research Triangle Park, NC; ECAO-R-092A.

Other EPA Documentation — U.S. EPA, 1987

Date of Agency Consensus — 11/25/97

To review the Summary of and Response to External Peer Review Comments, exit to the toxicological review, Appendix B (PDF).

A comprehensive review of toxicological studies published through June 2006 was conducted. No new health effects data were identified that would be directly useful in the revision of the existing RfD for Methyl methacrylate and a change in the RfD is not warranted at this time. For more information, IRIS users may contact the IRIS Hotline at hotline.iris@epa.gov or (202) 566-1676.

__I.A.7. EPA Contacts (Oral RfD)

Please contact the IRIS Hotline for all questions concerning this assessment or IRIS, in general, at (202)566-1676 (phone), (202)566-1749 (FAX), or hotline.iris@epa.gov (internet address).

_I.B. Reference Concentration for Chronic Inhalation Exposure (RfC)

Substance Name — Methyl methacrylate
CASRN — 80-62-6

Last Revised — 03/02/98

I.B.1. Inhalation RfC Summary

Critical Effect	Exposures*	UF	MF	RfC
Degeneration/atrophy of olfactory epithelium (male rats) Rat chronic inhalation study Hazelton Laboratories 1979a; Lomax, 1992; Lomax et al., 1997	BMC_{10}: 35 ppm BMC_{10}(ADJ): 25.6 mg/m^3 BMC_{10}(HEC): 7.2 mg/m^3	10	1	7E-1 mg/m^3

*Conversion Factors and Assumptions — The concentration associated with a 10% increased incidence (or extra risk) in the critical effect was determined using two dose-response functions. The 95% confidence limit on the concentration causing this benchmark response (BMC_{10}) was estimated to be 35 ppm (polynomial regression model). Assuming 25 °C and 760 mmHg and a molecular weight of 100.11, BMC_{10} (mg/m^3) = 35 ppm × 100.11/24.45 = 143 mg/m^3. BMC_{10}(ADJ) = 143 mg/m^3 × 6 h/24 h/day × 5 days/7 days = 25.6 mg/m^3. The BMC_{10}(HEC) was calculated for a gas:respiratory effect in the extrathoracic region. MVa = 0.25 L/min, MVh = 13.8 L/min, Sa(ET) = 11.6 cm^2, Sh(ET) = 177 cm^2. RGDR = (MVa/Sa)/(MVh/Sh) = 0.28. BMC(HEC) = 25.6 × RGDR = 7.2 mg/m^3.

I.B.2. Principal and Supporting Studies (Inhalation RfC)

Hazelton Laboratories America, Inc. (1979a). A two-year vapor inhalation safety evaluation study in rats: methyl methacrylate, final report. Vienna, VA: Hazleton Laboratories America, Inc.; project no. 417-354.

Lomax, LG. (1992) Histopathologic evaluation of the nasal cavities from Fisher 344 rats exposed to methyl methacrylate vapor for two years. Spring House, PA: Rohm and Haas Company.

Lomax, LG; Krivanek, N; Frame, SR. (1997) Chronic inhalation toxicity and oncogenicity of methyl methacrylate in rats and hamsters. Food Chem Toxicol 35:393-407.

F344 rats (70 of each sex per group) were exposed to mean concentrations of 0, 25, 99.79, or 396.07 ppm (0, 102.4, 408.6, 1,621.7 mg/m^3) for 6 h/day, 5 days/week (duration adjusted to 0, 18.3, 73, 289.6 mg/m^3) for 2 years (Hazelton Laboratories 1979a). No consistent trend with exposure was revealed, but microscopic examination of nasal tissues revealed minimal to slight focal rhinitis in 4/10 females exposed to 396.07 ppm (compared with 1 male and 1 female in the control group), and an inflammatory exudate was observed in 3 of the 4 exposed females. At 52 weeks, livers of 9/10 males and 6/10 females exposed to 396.07 ppm showed minimal nonsuppurative pericholangitis (compared with 5/10 control males and 2/10 control females). An increased incidence in lesions of mild rhinitis was observed in the nasal turbinates of exposed animals at week 104. These consisted of serous and purulent exudates, pleocellular infiltrates, distended submucosal glands, focal squamous metaplasia, and inflammatory polyps. Because the increased incidence was found in all exposure groups and did not appear to be concentration-dependent, these lesions may not have been treatment-related.

At the request of EPA, the U.S. Methacrylate Producers Association (MPA) commissioned a reexamination of the nasal tissue block and a rereview of the histopathology of the rat nasal tissues from the Hazelton (1979a) study (Lomax, 1992; Lomax et al., 1995). This reevaluation was requested because the initial study did not involve examination of the nasal tissues of the

low- and mid-exposure groups. In addition, because of MMA's propensity to cause effects in the olfactory epithelium as demonstrated in other studies (NTP, 1986), this reanalysis included examination of nasal tissue blocks in accordance with contemporary techniques with prescribed levels of sectioning. This reanalysis confirmed that chronic exposure to MMA does not appear to effect squamous epithelium at any exposure level. Effects in the respiratory epithelium were observed primarily at the 400 ppm exposure level, and were described as hyperplasia of submucosal glands and/or goblet cells in the anterior regions of the nasal cavity, especially around the dorsal meati and along the nasal septa. Inflammation of the mucosa and /or submucosa was also observed. Changes to respiratory epithelium were bilateral and slight to moderate in severity. Rats exposed to 100 or 400 ppm MMA had concentration-dependent histopathological changes to the olfactory portion of the dorsal meatus in the anterior portions of the nasal cavity. Microscopic changes were primarily observed in the olfactory region lining the dorsal meatus in the anterior region of the nasal cavity. These changes were characterized by degeneration and atrophy of the neurogenic epithelium and submucosal glands lining the dorsal meatus, basal cell hypoplasia, replacement of olfactory epithelium with ciliate (respiratory-like) epithelium, and inflammation of mucosa and submucosa. These changes were generally bilateral in distribution and the severity of the lesions varied from minimal to slight at 100 ppm to slight to moderate at 400 ppm. One male rat from the 400 ppm exposure group showed severe olfactory degenerative effects (Lomax, 1992). One male rat from each of the 100 and 400 ppm exposure groups had a small solitary polypoid mass attached to the lateral wall of one side of the anterior nasal cavity. These masses were morphologically similar, consisting of differentiated pseudoglandular structures arising from the respiratory epithelium, and were diagnosed as polypoid adenomas. The male rat from the 100 ppm group with the adenoma had concurrent moderate chronic inflammation of the nearby respiratory epithelium. Two male rats exposed to 400 ppm MMA had squamous metaplasia of the respiratory epithelium in the anterior region of the nasal cavity.

The hydrolysis of MMA by carboxylesterase enzymes and subsequent release of methacrylic acid in the olfactory tissue (Morris and Frederick, 1995) is likely the cause of the cytotoxicity in the olfactory region. Though it has been suggested that MMA metabolism is a detoxifying mechanism following oral exposure (Bereznowski, 1995), the metabolite, methacrylic acid, appears to be the toxic moiety in the olfactory tissues (Morris and Frederick, 1995; Lomax et al., 1995). In support of this assumption, the localization and activity of the metabolic enzyme, carboxylesterase, correlates quite well with the localization and severity of nasal lesions in rodents following MMA exposure (i.e., both occur predominantly in the olfactory epithelium and not respiratory epithelium) (Dahl et al., 1987; Bogdanffy et al., 1987; Bogdanffy, 1990; Frederick et al., 1994). Further, similar toxicity from compounds that metabolize to acids via the same metabolic route has been seen with ethyl acrylate (Miller et al., 1985), methyl and butyl acrylate (Klimisch, 1984), dibasic esters (Keenan et al., 1990), and glycol ether acetates (Miller et al., 1984), and exposures to acrylic and acetic acids directly have also caused similar olfactory- specific lesions (Miller et al., 1981; Stott and McKenna, 1985).

A polynomial mean response regression model (THRESH, I.C.F. Kaiser, 1990a) and a Weibull power mean response regression model (THRESHW, I.C.F. Kaiser, 1990b) were used to fit data from Lomax (1992) and Lomax et al. (1995) by the maximum likelihood method. These models were developed for use with dichotomous (incidence) data, and can either calculate a response threshold (for circumstances in which it is appropriate to presume the existence of an exposure level below which there is no response) or assign a threshold of zero (for circumstances in which it is appropriate to presume that all exposure levels emit a response). Because the mechanism for MMA olfactory toxicity is not well understood, the conservative model assumption of no threshold was employed. These models also provide the option of assuming a zero or nonzero background response. The only effect noted in control animals was minimal basal cell hyperplasia (5/39 control animals). For the purpose of calculating a BMC, it appears reasonable to assume a zero background for slight, moderate, and severe olfactory lesions.

Minimal lesions were excluded from the BMC analysis and a zero background was assumed. Using these criteria, the two models were applied to incidence data reported by Lomax (1992) and Lomax et al. (1995) for observed olfactory lesions in male and female rats.

Data for degeneration/atrophy of olfactory epithelium in males (0/39, 0/47, 35/48, and 38/38) were chosen for the derivation of the RfC because the concentration-response curves generated by both THRESH and THRESHW models were similar and of reasonable goodness of fit (*p* values = 0.616 and 0.768, respectively), and the resultant BMC values were lower than the BMCs for replacement by ciliated epithelium, the only other endpoint for which good model fit could be reached. An EPA review of benchmark analysis performed for several upper respiratory toxicants indicates that both the BMC values for the 5% and the 10% benchmark response (BMR) levels for a given endpoint generally fall between the NOAEL and the LOAEL for that endpoint (Gift, 1996). The benchmark response (BMR) chosen for use in the RfC derivation was a 10% increase in the incidence of a slight, moderate, or severe lesion. The 10% response level was chosen because of its closer proximity to the actual experimental data and because of the overall mild severity of the effect. The RfC is based on the BMC_{10}, which is the lower 95% confidence bound on the maximum likelihood estimate (MLE) of the concentration that causes a 10% increased incidence of this lesion. The two model predictions for the BMC_{10} from degeneration/atrophy of male rat olfactory epithelium were virtually identical, 39 (Weibull) and 35 (polynomial) ppm. The 35 ppm (143 mg/m^3) value was chosen for use in the RfC calculation because it results in a slightly more environmentally protective RfC. This value is slightly above the 25 ppm NOAEL and well below the 100 ppm LOAEL for degeneration/atrophy and inflammation. Details of the BMC_{10} derivation for this data set (model used, input assumptions, etc.) are provided in the IRIS support document for this compound.

When the BMC_{10}(mg/ m^3) is derived from a study in which laboratory animals are exposed intermittently (e.g., 6 h/day, 5 days/week), an adjustment is usually applied to account for the fact that the RfC is to protect against the worst-case scenario, continuous exposures. However, the EPA guidelines (EPA, 1994) recognize that, depending on the mechanism of action, such duration adjustment may not always be appropriate. In the case of acrylic acid, a compound that causes similar olfactory damage, there is information to suggest that a limited C × T relationship of exposure to toxic effects is operative over the course of at least the first 2 weeks of exposure at concentrations that cause minimal to moderate, reversible (if exposure is discontinued) olfactory effects (Lomax et al., 1994). The lack of lesions in rats after 28 days of exposure to 100 ppm MMA (Green, 1996), combined with the presence of lesions in rats following chronic (2-year) exposure to 100 ppm MMA (Lomax et al., 1997), suggests that these effects can progress with increased exposure duration. Thus, it is reasonable to suggest that continuous exposure to MMA could result in effects at concentrations below the NOAEL of an intermittent exposure study, and that the application of an adjustment factor to account for this is appropriate. Thus, the BMC_{10} of 143 mg/m^3 is adjusted to a BMC_{10}(ADJ) of 25.6 mg/m^3 (143 mg/m^3 × 6 h/24 h/day × 5 days/7 days = 25.6 mg/m^3). A human equivalent BMC_{10}, BMC_{10}(HEC), of 7.2 mg/m^3. is then calculated using default procedures for a gas:respiratory effect in the extrathoracic region [MVa = 0.25 L/min, MVh = 13.8 L/min, Sa(ET) = 11.6 cm^2, Sh(ET) = 177 cm^2. RGDR = (MVa/Sa)/(MVh/Sh) = 0.28. BMC(HEC) = 25.6 × RGDR = 7.2 mg/m^3], appropriate when peer-reviewed PBPK models are not available (US EPA, 1994).

I.B.3. Uncertainty and Modifying Factors (Inhalation RfC)

UF — 10.

A partial threefold uncertainty factor (UF) is applied to this effect level in consideration of possible intraspecies variation (UF_H; to protect sensitive human subpopulations). This UF is reduced from 10 because of extensive human occupational studies and case reports that consistently identify the irritant properties of MMA as the principal effect of concern from MMA

inhalation exposures. Little intraspecies variance is observed with respect to the identified critical effect, olfactory degeneration in laboratory animals (ECETOC, 1995; Lomax et al., 1997), and there is no reason to expect a high degree of intrahuman variability from this type of effect. Although Pickering et al. (1986) reported delayed asthmatic response following challenge with MMA, which would suggest that MMA is a possible respiratory sensitizer, no occupational studies identified MMA as a respiratory sensitizer. A partial intraspecies uncertainty factor of 3 is deemed sufficiently protective.

Two studies have noted increased resorptions in rats at 1,000 ppm exposures (Luo et al., 1986; ICI, 1977) and one did not (Solomon et al., 1993). However, the latter study was peer reviewed whereas Luo et al. (1986) was an abstract and ICI (1977) was an unpublished industry report. Multigenerational reproductive studies are not available for MMA; however, MMA is so reactive at the portal of entry that the potential for systemic effects is deemed remote. The observation of a portal-of-entry effect is consistent across both the oral and inhalation routes of exposure. Given these considerations, no uncertainty factor is applied to the RfC for database deficiencies.

A partial threefold uncertainty factor is used for interspecies extrapolation to account for potential toxicodynamic differences between rats and humans. This concern for potential toxicodynamic differences is warranted given the fact that humans may be less capable of recovering from olfactory damage than rats. "Rapid potentially anatomically correct recovery after massive destruction" is observed in rats when underlying basal cells are not damaged (Youngentob, 1997) and small islands of intact olfactory epithelium are "sufficient to allow for olfactory function" (Wong et al., 1997). In humans, it has been reported that patients with relatively mild to moderate olfactory damage fail to recover olfaction and "...even when basal cells remain intact, differentiating cells developing from them do not mature into receptor cells but can develop into squamous cells..." (Yamagishi and Nakano, 1992).

An attempt was made to account for toxicokinetic differences between the rat and human in the derivation of BMC_{10}(HEC). The HEC calculation attempts to account for the morphologic interspecies differences in the species as reflected by the different ratio of normal minute volume to surface area in rats versus humans. While, there remain several differences between rats and human that are not accounted for, most of these differences suggest that rat nasal passages are likely to be affected at lower MMA concentrations than those of humans. Most evidence suggests that the main metabolite of MMA, methacrylic acid, is the toxic moiety of concern (Lomax et al., 1997; Bereznowski, 1995; Morris and Frederick, 1995; ECETOC, 1995). Studies of carboxylesterase metabolic rates suggest that humans metabolize MMA in blood (Bereznowski, 1995) and in olfactory tissue (Mattes and Mattes, 1992; Greene, 1996) at a slower rate than rats, though at a slightly faster rate in the liver (Greene, 1996). In addition, rats are obligate nose breathers, whereas humans can breathe through the mouth during exertion and to avoid overpowering odors. EPA is aware of PBPK models for MMA (developed for the Methacrylate Producers Association by Andersen et al., 1996) and other acrylates (Morris and Frederick, 1995; Bogdanffy and Taylor, 1993) that should eventually help to reduce uncertainty in the quantification of these differences. The use of a PBPK model to update this assessment will be considered when EPA has completed its analysis of these various model approaches. In the meantime, a majority of the dosimetric/toxicokinetic evidence currently available suggests that humans would not be more sensitive than rats on this basis and that further reduction of the BMC_{10}(HEC) to account for interspecies dosimetric/toxicokinetic uncertainty is not necessary.

MF — 1.

I.B.4. Additional Studies/Comments (Inhalation RfC)

A. SUPPORTING STUDIES

The absorption and hydrolysis of MMA to methacrylic acid and subsequent metabolism via physiological pathways results in a low systemic toxicity by any route of exposure. However, 10% to 20% of inhaled MMA is deposited in the upper respiratory tract of rats and the hydrolysis of MMA by local nasal tissue esterases to methacrylic acid in this region has been cited as the primary reason for MMA's selective olfactory toxicity (Lomax, 1992; Lomax et al., 1997).

The EPA Toxicological Review for MMA summarizes key subchronic and chronic laboratory animals and human studies of MMA. Subchronic and chronic exposure of rats and mice to MMA by oral and inhalation routes (as well as dermal) results in effects consistent with its irritant properties. In inhalation studies, dose-related lesions have been observed in the upper respiratory tract, including rhinitis, inflammation associated with necrosis, degeneration/loss of olfactory epithelium in the nasal turbinates, and lung congestion. Exposures to very high levels of MMA (>1,000 ppm) can result in neurochemical and behavioral changes, reduced body weight gain, and degenerative and necrotic changes in the liver, kidney, brain, spleen, and bone marrow. Relatively low concentrations can cause changes in liver enzyme activities. The data concerning MMA's ability to cause cardiovascular effects are inconsistent. Several publications in the literature suggest that MMA may have cardiovascular and/or neurotoxic effects in occupationally exposed human beings. These effects may not represent neurotoxicity, as they are generally nonspecific and workers were exposed to several other toxic compounds. In general, MMA has not resulted in serious adverse effects to humans. In certain individuals it has been shown to induce allergic dermatitis from skin contact. Mild eye irritation and respiratory tract irritation have been reported, but the evidence available does not allow for a determination regarding respiratory sensitization.

Evidence for developmental effects from inhalation exposure is mixed and generally occurred at maternally toxic exposure levels. Solomon et al. (1993) found no developmental effects in rats exposed 6 h/day during days 6-15 of gestation to atmospheric concentrations of up to 2,028 ppm (8,304 mg/m^3). Tansy (1979) and McLaughlin et al. (1978) found no developmental effects in mice exposed 6 h/day to up to 400 ppm and 2 h/day to 1,330 ppm, respectively, during days 6-15 of gestation. However, Nicholas et al. (1979) found evidence of developmental effects (early fetal deaths, delayed ossification, decreased fetal body weight and crown-rump length, hematomas) in Sprague-Dawley rats exposed for approximately 1 h/day during days 6-15 of gestation to levels more than an order of magnitude higher (110,000 mg/m^3). However, nearly 20% of the exposed pregnant rats died at this exposure level. In addition, ICI (1977) and Luo et al. (1986) describe both delayed ossification and increased resorptions in rats exposed during days 6-15 of gestation to 1,000 ppm MMA (5 h/day and 2 h/3 days, respectively). No adequate one- or two-generation reproductive studies were available by any route of exposure. MMA did not reveal an effect on male fertility in mice inhaling up to 9,000 ppm MMA for 6 h/day over a period of 5 days (ICI, 1976). These data suggest that at high, maternally toxic doses, MMA can cause developmental effects. However, there is no reason to believe that developmental toxicity should represent a critical or co-critical effect in the RfC or RfD derivation. The lack of adequate reproductive studies is not a major concern given the limited evidence for systemic or genotoxic effects from MMA exposure, but has been considered in the determination of uncertainty factors.

For more detail on other Hazard Identification Issues, exit to the toxicological review, Section 4.7 (PDF)

I.B.5. Confidence in the Inhalation RfC

Study — High
Database — Medium to high
RfC — Medium to high

The overall confidence in this RfC assessment is medium to high. The RfC is based on a long-term rat inhalation study (Hazelton Laboratories, Inc., 1979a) performed with relatively large group sizes in which, with additional investigations (Lomax, 1992; Lomax et al., 1995), thorough histopathologic analyses were performed on all relevant tissues. What is considered to be the primary target organ, the nasal passage, was particularly well described, and the study was able to identify both a NOAEL and a LOAEL. The scientific quality of the combined Hazelton Laboratories (1979a) and subsequent reanalyses (Lomax, 1992; Lomax et al., 1995) is high.

The confidence in the inhalation database available for MMA is rated as medium to high. Acceptable developmental studies were carried out in two species, rats and mice, with effects only observed in offspring at levels more than 10-fold higher than the LOAEL for the chosen critical (olfactory) effect. Multigenerational reproductive studies are not available for MMA. However, protection against the portal-of-entry effects observed at low exposure levels across both the oral and inhalation routes of exposure is deemed likely to also protect against any possible multigenerational reproductive effects. Given these considerations the inhalation database and the RfC are given medium to high confidence.

EPA recognizes that PBPK models are under development for MMA (Andersen et al., 1996) and other acrylates (Morris and Frederick, 1995; Bogdanffy and Taylor, 1993). The results of these ongoing investigations are under review by the Agency and are expected to help increase confidence in the estimation of a human equivalent concentration and clarify the different species sensitivities.

For more detail on Characterization of Hazard and Dose Response, exit to the toxicological review, Section 6 (PDF).

I.B.6. EPA Documentation and Review of the Inhalation RfC

Source Document — This assessment is presented in the Toxicological Review of Methyl Methacrylate (CAS No. 80-62-6). (EPA, 1998).

U. S. Environmental Protection Agency. (1985) Health and environmental effects profile for methyl methacrylate. Cincinnati, OH: Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office; report no. EPA/600/X-85/364. Available from: NTIS, Springfield, VA; PB88-178785/XAB.

U.S. Environmental Protection Agency. (1988) Health and environmental effects profile for methyl methacrylate. NTIS/PB88-178785.

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Agency Consensus Review Date -- 11/25/97

To review the Summary of and Response to External Peer Review Comments, exit to the toxicological review, Appendix B (PDF)

A comprehensive review of toxicological studies published through June 2006 was conducted. No new health effects data were identified that would be directly useful in the revision of the existing RfC for Methyl methacrylate and a change in the RfC is not warranted at this time. For more information, IRIS users may contact the IRIS Hotline at hotline.iris@epa.gov or (202) 566-1676.

__I.B.7. EPA Contacts (Inhalation RfC)

Please contact the IRIS Hotline for all questions concerning this assessment or IRIS, in general, at (202)566-1676 (phone), (202)566-1749 (FAX), or hotline.iris@epa.gov (internet address).

_II. Carcinogenicity Assessment for Lifetime Exposure

Substance Name — Methyl methacrylate
CASRN — 80-62-6
Last Revised — 03/02/98

Section II provides information on three aspects of the carcinogenic assessment for the substance in question; the weight-of-evidence judgment of the likelihood that the substance is a human carcinogen, and quantitative estimates of risk from oral exposure and from inhalation exposure. The quantitative risk estimates are presented in three ways. The slope factor is the result of application of a low-dose extrapolation procedure and is presented as the risk per (mg/kg)/day. The unit risk is the quantitative estimated in terms of either risk per ug/L drinking water or risk per ug/m^3 air breathed. The third form in which risk is presented is a drinking water or air concentration providing cancer risks of 1 in 10,000, 1 in 100,000, or 1 in 1,000,000. The rationale and methods used to develop the carcinogenicity information in IRIS are described in the Risk Assessment Guidelines of 1986 (EPA/600/8-87/045) and in the IRIS Background Document. IRIS summaries developed since the publication of EPA's more recent Proposed Guidelines for Carcinogen Risk Assessment also utilize those Guidelines where indicated (Federal Register 61(79):17960-18011, April 23, 1996). Users are referred to Section I of this IRIS file for information on long-term toxic effects other than carcinogenicity.

_II.A. Evidence for Human Carcinogenicity

__II.A.1. Weight-of-Evidence Characterization

Under EPA's 1986 Guidelines for Carcinogen Risk Assessment, MMA would be classified as *evidence of non-carcinogenicity for humans* or a Group E chemical. Under the Proposed Guidelines for Carcinogenic Risk Assessment (U.S. EPA, 1996), MMA is considered *not likely to be carcinogenic to humans* by any route of exposure because it has been evaluated in four well-conducted chronic inhalation studies in three appropriate animal species without demonstrating carcinogenic effects.

Basis — The results of the 2-year inhalation studies conducted for NTP showed no evidence of carcinogenicity of MMA for male F344/N rats exposed at 500 or 1,000 ppm, for female F344/N rats exposed at 250 or 500 ppm, or for female B6C3F1 mice exposed at 500 or 1,000 ppm. In addition, no increase was seen in the number or type of tumors in either rats or hamsters from the chronic inhalation study performed by Hazelton Laboratories (1979a,b). No carcinogenic activity was reported in a chronic oral study (Borzelleca et al., 1964). Fewer animals were used and the experimental protocal and results of this oral study were not as well documented as for

the inhalation study. However, acute oral exposure studies and structure-activity relationship comparisons with other acrylates suggest that the introduction of a methyl group to the acrylate moiety (e.g., EA to MMA) negates carcinogenic activity. Epidemiology studies show no clear excess of cancer. Though a report suggesting increased colon cancer among ethyl acrylate/MMA- exposed workers exists, a high background for this effect has been documented for the location and time of this study, the effects were not reproduced in other similar and more recent studies, a clear relationship between exposure and effect was not demonstrated, and the extent that ethyl acrylate concurrent exposure confounded results could not be determined. Given these structure-activity relationship considerations, the low potential for cancer from MMA exposure indicated in genotoxicity, laboratory animal and epidemiology studies suggests that MMA does not represent a carcinogenic hazard to humans.

For more detail on Characterization of Hazard and Dose Response, exit to the toxicological review, Section 6 (PDF).

For more detail on other Hazard Identification Issues, exit to the toxicological review, Section 4.7 (PDF)

II.A.2. Human Carcinogenicity Data

Inadequate. Limited epidemiological data are available to determine whether the incidence of various malignancies is higher in groups occupationally exposed to MMA versus those not exposed, and no studies have been reported on whether or not smoking is a related factor in the occurrence of malignancies in MMA-exposed workers. One retrospective epidemiological study that relates to malignancies was conducted at the Bristol Plant, PA, which manufactures plastics, leather chemicals, etc. (Monroe, 1984; Walker et al., 1991). In this study of Bristol Plant employees hired prior to 1946 (Early Bristol cohort), an excess of cancer of the large intestine and rectum was noted. However, an increase in these types of cancers was not observed in similar populations at separate sites, and in subsequent evaluations of the same site (Walker et al., 1991; ECETOC, 1995; Collin et al, 1989). Collins et al. (1989) have noted that during the 1970's, the county in which the plant was located had a high colorectal cancer rate, at the 75th percentile for the United States.

Some evidence of an increased death rate from cancer and noncancer respiratory disease is provided by the American Cyanamid (Collins et al., 1989) and Knoxville (Walker et al., 1991) cohorts. However, in both of these cohorts, exposure to MMA was considerably lower than in the Early Bristol cohort, which showed no such excess. Others have suggested that these increases were lifestyle related (ECETOC, 1995).

Some instances of possible association of human neoplasms with MMA have been reported, but most have been clearly associated with polymethyl methacrylate. Wines (1973) reported on a patient who developed bladder carcinoma adjacent to intrapelvic cement (polymethyl methacrylate) following a Charnley total hip replacement; Thompson and Entin (1969) reported on the occurrence of a chondrosarcoma intimately associated with the fibrous capsule surrounding lucite (polymethacrylate) spheres used as plombage for compressing a tuberculous cavity; Routledge (1973) described a case of granuloma of the upper lobe of the left lung in a worker in a hospital department making polymethacrylate contact lenses.

II.A.3. Animal Carcinogenicity Data

No Evidence. Carcinogenic tests have been performed which suggest that tumors can form when laboratory animals are subjected to subcutaneous implants of poly-MMA (Laskin et al., 1954; Ferguson, 1977). While some researchers (Homsy et al., 1972, Bright et al., 1972) have shown some leaching of monomeric MMA from poly-MMA surgical implants, Ferguson (1977)

suggests that sarcomas that arise following subcutaneous implants of poly-MMA can be attributed to mechanical processes involving topographic interaction of the solid surface with normal cells, especially macrophages. In the experiments of Oppenheimer et al. (1955), no tumors were induced when monomeric MMA was applied dermally to the back of the neck of rats. While suggestive with respect to whether mode of application has bearing on the results of such experiments, the Oppenheimer study should not be considered sufficient for evaluating the carcinogenic potential of MMA, as the exposure period was just 4 mo and only 10 animals were tested.

In the studies by Hazelton Laboratories (1979a,b) Fischer 344 rats and Charles River Lakeview Golden Hamsters were exposed to MMA vapors at 0, 25, 100, and 400 ppm for 6 h/day for 5 days/week for 2 years and 18 mo, respectively. No increase was seen in the number or type of tumors in either rats or hamsters, indicating that MMA was not carcinogenic in these two species under those conditions. Appearance of a polypoid adenoma in the nasal cavity of two MMA-exposed male rats (Lomax, 1992) is not likely to be associated with MMA exposure, and these benign neoplasms have been reported in control rats as well. Similarly, a 2-year NTP inhalation bioassay in rats and mice gave negative results for carcinogenicity, although the animals may not have been tested at the maximum tolerated dose (National Toxicology Program, 1986; Chan et al., 1988).

Borzelleca et al. (1964) found no significant toxic effects in male and female dogs (2 males and 2 females per treatment group) receiving MMA via gelatin capsule in the diet at 10, 100, or 1,473 ppm daily for 1 year. The high exposure concentration represented a time-weighted average based on the 1,000 ppm value increasing to 1,200 ppm at five weeks, to 1,400 ppm at seven weeks, and to 1,500 ppm at nine weeks.

Borzelleca et al. (1964) also exposed groups of 25 male and 25 female Wistar rats to MMA in drinking water for 104 weeks. The initial exposure concentrations were 6, 60, and 2,000 ppm MMA. The low and medium exposures were increased to 7 and 70 ppm, respectively, at the start of the fifth month, resulting in TWA exposure concentrations of 6.85 and 68.46 ppm MMA. Survival of exposed rats was not significantly different from controls. An initial reduction in body weight gain was observed in both males and females exposed to 2,000 ppm MMA, which reverted to control levels by week 3 (females) and week 6 (males). This is likely the result of reported reduced food intake during the first month, which was not observed in the second month and beyond. Tissues examined included heart, lung, liver, kidney, urinary bladder, spleen, gastroenteric, skeletal, muscle, skin, brain, thyroid, adrenal, pancreas, pituitary, and gonads. The only effect observed was an increased kidney/body-weight ratio in female rats exposed to 2,000 ppm MMA. No abnormalities or lesions related to MMA were identified from histopathological examination of the tissues of exposed rats.

__II.A.4. Supporting Data for Carcinogenicity

When tested at cytotoxic concentrations, MMA does not appear to be mutagenic to bacteria (National Toxicology Program, 1986; ECETOC, 1995; Waegemaekers and Bensink, 1984). MMA has been shown to be an in vitro clastogen in mammalian cell gene mutation and chromosomal aberration assays (National Toxicology Program, 1986; ECETOC, 1995). However, MMA has not been shown to result in clastogenic effects or dominant lethal mutations following laboratory animal in vivo inhalation (ICI, 1976a) or oral exposures (Hachiya et al., 1981), and reports of chromosomal damage from in vivo human data (Marez et al., 1991; Seji et al., 1994) are equivocal.

_II.B. Quantitative Estimate of Carcinogenic Risk from Oral Exposure

No data available.

II.C. Quantitative Estimate of Carcinogenic Risk from Inhalation Exposure

No data available.

II.C.1. Summary of Risk Estimates

II.C.1.1. Unit Risk

No data available.

II.C.1.2. Extrapolation Method

No data available.

II.C.2. Dose-Response Data for Carcinogenicity, Inhalation Exposure

No data available.

II.C.3. Additional Comments (Carcinogenicity, Inhalation Exposure)

Acrylic acid, four monofunctional acrylates, eight polyfunctional (di- or tri-) acrylates, a dimethacrylate, and a trimethacrylate have been tested in skin-painting cancer bioassays. Acrylic acid, 2-ethylhexyl acrylate, and three diacrylates caused skin tumors. Methyl acrylate (MA), ethyl acrylate (EA), n-butyl acrylate (BA), and methyl methacrylate have been tested in chronic inhalation bioassays and found to be negative with respect to carcinogenicity (Woo et al., 1988). While the Borzelleca et al. (1964) drinking water studies did not report carcinogenicity for either EA or MMA exposure, EA was found to cause forestomach tumors following gavage exposure (NTP, 1983). However, the fact the EA has been found to cause forestomach tumors at high gavage doses (NTP, 1983) does not necessarily implicate MMA. This is suggested by structure-activity relationship studies that demonstrate that the addition of a methyl group to the acrylate moiety tends to abolish carcinogenic activity (Woo et al., 1988) and gavage dosing of analogues of EA demonstrating that the forestomach toxicity required the intact molecule (an ester moiety, the double bond, and no substitution at carbon number 2) (Ghanayem et al., 1985). In another paper, Ghanayem et al. (1986) reported that cell proliferation of the rat forestomach (believed to be a precursor effect to tumors caused by this compound) was apparent in all rats (12/12) following 2-week gavage administration of EA at both 100 and 200 mg/kg, but was not apparent in any rats exposed to 100 mg/kg MMA (0/8) and in just 1/8 rats exposed to 200 mg/kg MMA. This latter increase was not statistically significant and the effect was much less severe than the effects caused by EA at either dose. Thus, structure-activity relationship analysis does not suggest that MMA would be carcinogenic by any route.

II.C.4. Discussion of Confidence (Carcinogenicity, Inhalation Exposure)

Although some cases of sarcomas have been reported following implants of poly-MMA, it is likely that these are the result of mechanical processes involving topographic interaction of the solid surface with normal cells and are not due to leaching of monomeric MMA from poly-MMA surgical implants. The results of the 2-yr inhalation studies conducted for NTP showed no

evidence of carcinogenicity of MMA for male F344/N rats exposed at 500 or 1,000 ppm, for female F344/N rats exposed at 250 or 500 ppm, or for female B6C3F1 mice exposed at 500 or 1,000 ppm. In addition, no increase was seen in the number or type of tumors in either rats or hamsters from the chronic inhalation study performed by Hazelton Laboratories (1979a,b). Appearance of a polypoid adenoma in the nasal cavity of two MMA exposed male rats (Lomax, 1992) is not likely to be associated with MMA exposure, and these benign neoplasms have been reported in control rats as well.

_II.D. EPA Documentation, Review, and Contacts (Carcinogenicity Assessment)

__II.D.1. EPA Documentation

Source Document — This assessment is presented in the Toxicological Review of Methyl Methacrylate (CAS No. 80-62-6). (EPA, 1998).

This assessment was peer reviewed by external scientists. Their comments have been evaluated carefully and incorporated in finalization of this IRIS Summary. A record of these comments is included as an appendix to Toxicological Review of Methyl Methacrylate (MMA) in support of summary information on Integrated Risk Information System (IRIS). ***To review this appendix, exit to the toxicological review, Appendix B, Summary of and Response to External Peer Review Comments (PDF)***

__II.D.2. EPA Review (Carcinogenicity Assessment)

Agency Consensus Date — 11/25/97

A comprehensive review of toxicological studies published through June 2006 was conducted. No new health effects data were identified that would be directly useful in the revision of the existing carcinogenicity assessment for Methyl methacrylate and a change in the assessment is not warranted at this time. For more information, IRIS users may contact the IRIS Hotline at hotline.iris@epa.gov or (202)566-1676.

__II.D.3. EPA Contacts (Carcinogenicity Assessment)

Please contact the IRIS Hotline for all questions concerning this assessment or IRIS in general at (202)566-1676 (phone), (202)566-1749 (FAX), or hotline.iris@epa.gov (Internet address).

_III. [reserved]
_IV. [reserved]
_V. [reserved]

_VI. Bibliography

Substance Name — Methyl methacrylate
CASRN — 80-62-6
Last Revised — 03/02/98

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_VII. Revision History

Substance Name — Methyl methacrylate
CASRN — 80-62-6

Date	Section	Description
004/01/1997	III., IV., V.	Drinking Water Health Advisories, EPA Regulatory Actions, and Supplementary Data were removed from IRIS on or before April 1997. IRIS users were directed to the appropriate EPA Program Offices for this information.
03/02/98	I.A., I.B., II., VI.	New RfD, RfC, cancer assessments
12/03/2002	I.A.6., I.B.6., II.D.2.	Screening-Level Literature Review Findings message has been added.
07/05/2006	I.A.6., I.B.6., II.D.2.	Screening-Level Literature Review Findings message has been removed and replaced by comprehensive literature review conclusions.

_VIII. Synonyms

Substance Name — Methyl methacrylate
CASRN — 80-62-6
Last Revised — 03/02/98

- Methacrylic acid, methyl ester
- Methacrylate monomer
- Methyl a-methylacrylate
- Methyl 2-methyl-2-propenoate

IRIS Home

Chronic Health Hazards for Non-Carcinogenic Effects

Reference Dose for Chronic Oral Exposure (RfD)

- Oral RfD Summary
- Principal and Supporting Studies
- Uncertainty and Modifying Factors
- Additional Studies/Comments
- Confidence in the Oral RfD
- EPA Documentation and Review

Reference Concentration for Chronic Inhalation Exposure (RfC)

- Inhalation RfC Summary
- Principal and Supporting Studies
- Uncertainty and Modifying Factors
- Additional Studies/Comments
- Confidence in the Inhalation RfC
- EPA Documentation and Review

Carcinogenicity Assessment for Lifetime Exposure

Evidence for Human Carcinogenicity

- Weight-of-Evidence Characterization
- Human Carcinogenicity Data
- Animal Carcinogenicity Data
- Supporting Data for Carcinogenicity

Quantitative Estimate of Carcinogenic Risk from Oral Exposure

- Summary of Risk Estimates
- Dose-Response Data
- Additional Comments

Appendix E

1,2,2-Trichloro-1,1,2-Trifluoroethane Calculations and IRIS Data

Additional Trichloro-1,2,2-trifluoroethane, 1,1,2- RSR Criteria Calculations

Input Parameters

Direct Exposure Criteria Parameters			
RISK	Target Cancer Risk Level	Unitless	0.000001
HI	Hazard Index	Unitless	1
RFD	Reference Dose (From IRIS)	$(mg/kg\text{-}day)^{-1}$	3.000
IR	Ingestion Rate	mg/day	50
EF	Exposure Frequency	days/year	250
ED	Exposure Duration	years	25
CF	Conversion Factor	kg/mg	0.000001
BW	Body Weight	kg	70
AT	Averaging Time, for carcinogens	days	25550
AT_A	Averaging Time, Adult non-carcinogens	days	9125
Pollutant Mobility Parameters			
RFD	Reference Dose (from IRIS)	$(mg/kg\text{-}day)^{-1}$	3.000
HI	Hazard Index	Unitless	1
BW	Body Weight	kg	70
AT	Averaging Time	days	25550
SA	Source Allocation	Unitless	0.2
IR	Ingestion Rate	l/day	2
EF	Exposure Frequency	days/year	365
ED	Exposure Duration	years	70
CF	Conversion Factor	Unitless	0.001

Calculations

Soil Matrix	**Value**	**Units**
Industrial/commercial direct exposure criteria (I/C DEC)		
DEC_{RB} = (RFD *HI)* ((BW*AT)/(IR*EF*ED*CF))	17169600	mg/kg
Groundwater Pollutant Mobility Criteria for GB Areas (GB PMC)		
GB PMC = GWPC * 20 (µg/L to µg/kg) * 10 (Conversion from GA to GB)	4200000	µg/kg
Groundwater Matrix		
Groundwater Protection Criteria (GWPC)		
GWPC = (RFD x HI) x ((BW x AT x SA) / (IR x EF x ED x CF))	21000	µg/L

Notes:

Reference Dose collected from EPA IRIS website at www.epa.gov/iris/ Accessed March 11, 2009

GWPC and I/C DEC formulas referenced to the Nov. 18, 2002 memo for CTDEP Corrected Criteria Formula

http://www.epa.gov/NCEA/iris/subst/0123.htm
Last updated on Thursday, January 10th, 2008.

Integrated Risk Information System

1,1,2-Trichloro-1,2,2-trifluoroethane (CFC-113) (CASRN 76-13-1)

view QuickView

MAIN CONTENTS

List of IRIS Substances

Search IRIS by Keyword

IRIS Summaries/Toxicological Reviews

Entire IRIS Website

Reference Dose for Chronic Oral Exposure (RfD)

0123

1,1,2-Trichloro-1,2,2-trifluoroethane (CFC-113); CASRN 76-13-1

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR CFC-113

File First On-Line 01/31/1987

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	on line	02/01/1996
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	no data	

_I. Chronic Health Hazard Assessments for Noncarcinogenic Effects

_I.A. Reference Dose for Chronic Oral Exposure (RfD)

Substance Name — 1,1,2-Trichloro-1,2,2-trifluoroethane (CFC-113)
CASRN — 76-13-1
Last Revised — 02/01/1996

The oral Reference Dose (RfD) is based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. It is expressed in units of mg/kg-day. In general, the

RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Please refer to the Background Document for an elaboration of these concepts. RfDs can also be derived for the noncarcinogenic health effects of substances that are also carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

NOTE: The Oral RfD for 1,1,2-trichloro-1,2,2-trifluoroethane may change in the near future pending the outcome of a further review now being conducted by the Oral RfD Work Group.

__I.A.1. Oral RfD Summary

Critical Effect	Experimental Doses*	UF	MF	RfD
Psychomotor impairment Epidemiologic Study: Human Occupational Exposure Imbus and Adkins, 1972	NOAEL: 5358 mg/cu.m converted to 273 mg/kg/day	10	1	3E+1 mg/kg/day

*Conversion Factors: 10 cu.m/day (8-hour human breathing volume), 5 days/7 days, 0.5 absorption factor, 70 kg bw; thus, 5358 mg/cu.m x 10 cu.m/day x 5 days/7 days x 0.5/70 kg = 273 mg/kg/day

__I.A.2. Principal and Supporting Studies (Oral RfD)

Imbus, H.R. and C. Adkins. 1972. Physical examination of workers exposed to trichlorotrifluoroethane. Arch. Environ. Health. 24(4): 257-261.

Several animal inhalation studies reported negative results in dogs, rabbits, and rats chronically exposed to very high concentrations of trichlorotrifluoroethane (U.S. EPA, 1983). No apparent adverse effects have been reported in humans occupationally exposed to trichlorotrifluoroethane at either 500 mg/cu.m levels for 11 years or 5358 mg/cu.m levels for 2.77 years (Imbus and Adkins, 1972).

Slight impairment of psychomotor performance was reported in male volunteers exposed to trichlorotrifluoroethane concentrations of 19,161 mg/cu.m for 2.75 hours (Stopps and McLaughlin, 1967). This exposure period was too brief to consider a NOAEL for chronic exposure. Therefore, the RfD of 30 mg/kg/day is considered protective.

__I.A.3. Uncertainty and Modifying Factors (Oral RfD)

UF — The uncertainty factor of 10 accounts for the expected interhuman variability to the toxicity of this chemical in lieu of specific data.

MF — None

__I.A.4. Additional Studies/Comments (Oral RfD)

None.

__I.A.5. Confidence in the Oral RfD

Study — Low
Database — Low
RfD — Low

Confidence in the chosen study, database, and RfD are all considered low. Despite the fact that the chosen study describes human data and the fact that several chronic studies in animals are supportive, uncertainties in both the exposure levels and route extrapolation preclude higher confidence ratings.

__I.A.6. EPA Documentation and Review of the Oral RfD

Source Docuemnt -- U.S. EPA, 1983

Other EPA Documentation — None

Agency Work Group Review — 06/24/1985, 07/08/1985

Verification Date — 07/08/1985

Screening-Level Literature Review Findings — A screening-level review conducted by an EPA contractor of the more recent toxicology literature pertinent to the RfD for 1,1,2-Trichloro-1,2,2-trifluoroethane conducted in September 2002 did not identify any critical new studies. IRIS users who know of important new studies may provide that information to the IRIS Hotline at hotline.iris@epa.gov or (202)566-1676.

__I.A.7. EPA Contacts (Oral RfD)

Please contact the IRIS Hotline for all questions concerning this assessment or IRIS, in general, at (202)566-1676 (phone), (202)566-1749 (FAX) or hotline.iris@epa.gov (internet address).

_I.B. Reference Concentration for Chronic Inhalation Exposure (RfC)

Substance Name — 1,1,2-Trichloro-1,2,2-trifluoroethane (CFC-113)
CASRN — 76-13-1

Not available at this time.

_II. Carcinogenicity Assessment for Lifetime Exposure

Substance Name — 1,1,2-Trichloro-1,2,2-trifluoroethane (CFC-113)
CASRN — 76-13-1

Not available at this time.

_III. [reserved]
_IV. [reserved]
_V. [reserved]

_VI. Bibliography

Substance Name — 1,1,2-Trichloro-1,2,2-trifluoroethane (CFC-113)
CASRN — 76-13-1
Last Revised — 01/01/1990

_VI.A. Oral RfD References

Imbus, H.R. and C. Adkins. 1972. Physical examination of workers exposed to trichlorotrifluoroethane. Arch. Environ. Health. 24(4): 257-261.

Stopps, G.J. and M. McLaughlin. 1967. Psychophysiological testing of human subjects exposed to solvent vapors. Amer. Ind. Hyg. Assoc. J. 28: 43-50.

U.S. EPA. 1983. Health Assessment Document for 1,1,2-trichloro-1,2,2- trifluoroethane (chlorofluorocarbon CFC 113). Office of Air Quality Planning and Standards, Research Triangle Park, NC. EPA-600/8-82-002F. NTIS PB84- 118843. (Final Report)

_VI.B. Inhalation RfC References

None

_VI.C. Carcinogenicity Assessment References

None

_VII. Revision History

Substance Name — 1,1,2-Trichloro-1,2,2-trifluoroethane (CFC-113)
CASRN — 76-13-1

Date	Section	Description
04/06/1987	I.A.1.	RfD corrected
03/01/1988	I.A.1.	Dose conversion factor corrected

12/01/1988	I.A.	RfD noted as pending change
01/01/1990	I.A.6.	Added U.S. EPA citation
01/01/1990	VI.	Bibliography on-line
06/01/1990	IV.A.1.	Area code for EPA contact corrected
01/01/1992	I.A.7.	Primary contact changed
01/01/1992	IV.	Regulatory actions updated
04/01/1992	IV.A.1.	CAA regulatory action withdrawn
08/01/1995	I.A.	EPA's RfD/RfC and CRAVE workgroups were discontinued in May, 1995. Chemical substance reviews that were not completed by September 1995 were taken out of IRIS review. The IRIS Pilot Program replaced the workgroup functions beginning in September, 1995.
02/01/1996	I.A.7.	Contact changed
04/01/1997	III., IV., V.	Drinking Water Health Advisories, EPA Regulatory Actions, and Supplementary Data were removed from IRIS on or before April 1997. IRIS users were directed to the appropriate EPA Program Offices for this information.
12/03/2002	I.A.6.	Screening-Level Literature Review Findings message has been added.

_VIII. Synonyms

Substance Name — 1,1,2-Trichloro-1,2,2-trifluoroethane (CFC-113)
CASRN — 76-13-1
Last Revised — 01/31/1987

- 76-13-1
- ARCTON 63
- ARKLONE P
- CFC-113
- DAIFLON S 3
- ETHANE, 1,1,2-TRICHLORO-1,2,2-TRIFLUORO-
- FLUOROCARBON 113
- FREON 113
- FREON 113TR-T
- FREON F113
- FREON TF
- FRIGEN 113a
- FRIGEN 113 TR-T
- GENETRON 113
- HALOCARBON 113
- ISCEON 113
- KHLADON 113
- R 113
- REFRIGERANT 113
- TRICHLOROTRIFLUOROETHANE
- 1,1,2-Trichloro-1,2,2-trifluoroethane
- Trichloro-1,2,2-trifluoroethane, 1,1,2-

- 1,1,2-TRIFLUORO-1,2,2- TRICHLOROETHANE
- UCON 113
- UCON 113/HALOCARBON 113
- UCON FLUOROCARBON 113

IRIS Home

Chronic Health Hazards for Non-Carcinogenic Effects

Reference Dose for Chronic Oral Exposure (RfD)

- Oral RfD Summary
- Principal and Supporting Studies
- Uncertainty and Modifying Factors
- Additional Studies/Comments
- Confidence in the Oral RfD
- EPA Documentation and Review

Reference Concentration for Chronic Inhalation Exposure (RfC)

- Inhalation RfC Summary
- Principal and Supporting Studies
- Uncertainty and Modifying Factors
- Additional Studies/Comments
- Confidence in the Inhalation RfC
- EPA Documentation and Review

Carcinogenicity Assessment for Lifetime Exposure

Evidence for Human Carcinogenicity

- Weight-of-Evidence Characterization
- Human Carcinogenicity Data
- Animal Carcinogenicity Data
- Supporting Data for

Carcinogenicity

Quantitative Estimate of Carcinogenic Risk from Oral Exposure

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- Dose-Response Data
- Additional Comments
- Discussion of Confidence

Quantitative Estimate of Carcinogenic Risk from Inhalation Exposure

- Summary of Risk Estimates
- Dose-Response Data
- Additional Comments
- Discussion of Confidence
- EPA Documentation, Review and, Contacts

Bibliography

Revision History

Synonyms

Appendix F

Benzyl Alcohol Calculations

Additional Benzyl alcohol RSR Criteria Calculations

Input Parameters

Direct Exposure Criteria Parameters			
RISK	Target Cancer Risk Level	Unitless	0.000001
HI	Hazard Index	Unitless	1
RFD	Reference Dose (From PPRTV)	$(mg/kg\text{-}day)^{-1}$	0.500
IR	Ingestion Rate	mg/day	50
EF	Exposure Frequency	days/year	250
ED	Exposure Duration	years	25
CF	Conversion Factor	kg/mg	0.000001
BW	Body Weight	kg	70
AT	Averaging Time, for carcinogens	days	25550
AT_A	Averaging Time, Adult non-carcinogens	days	9125
Pollutant Mobility Parameters			
RFD	Reference Dose	$(mg/kg\text{-}day)^{-1}$	0.500
HI	Hazard Index	Unitless	1
BW	Body Weight	kg	70
AT	Averaging Time	days	25550
SA	Source Allocation	Unitless	0.2
IR	Ingestion Rate	l/day	2
EF	Exposure Frequency	days/year	365
ED	Exposure Duration	years	70
CF	Conversion Factor	Unitless	0.001

Calculations

Soil Matrix	**Value**	**Units**
Industrial/commercial direct exposure criteria (I/C DEC)		
DEC_{RB} = (RFD *HI)* ((BW*AT)/(IR*EF*ED*CF))	2861600	mg/kg
Groundwater Pollutant Mobility Criteria for GB Areas (GB PMC)		
GB PMC = GWPC * 20 (µg/L to µg/kg) * 10 (Conversion from GA to GB)	700000	µg/kg
Groundwater Matrix		
Groundwater Protection Criteria (GWPC)		
GWPC = (RFD x HI) x ((BW x AT x SA) / (IR x EF x ED x CF))	3500	µg/L

Notes:

Reference Dose collected from EPA 2008 PPRTV(provisional peer reviewed toxicity value)

GWPC and I/C DEC formulas referenced to the Nov. 18, 2002 memo for CTDEP Corrected Criteria Formula

Key: I = IRIS; P = PPRTV; A = ATSDR; C = Cal EPA; H = HEAST; W = WHO; S = see user guide Section 5; L = see user guide on lead; M = mutagen; V = volatile; c = cancer; * = where: n SL < 100X c SL; ** = where n SL < 10X c SL; n = noncancer; m = Concentration may exceed ceiling limit (See User's Guide); s = Concentration may exceed Csat (See User's Guide); SSL values are based on DAF=1

Contaminant		Toxicity and Chemical-specific Information											Screening Levels										Protection of Groundwater	
Analyte	CAS No.	SFO (mg/kg-day)$^{-1}$	key	IUR (ug/m^3)$^{-1}$	key	RfDo (mg/kg-day)	key	RfCi (mg/m^3)	key	voc	mutagen	RAGS Part E GIABS	RAGS Part E ABS	Csat mg/kg	Residential Soil mg/kg	key	Industrial Soil mg/kg	key	Residential Air ug/m^3	key	Industrial Air ug/m^3	key	Tapwater ug/L	key	MCL ug/L	Risk-based SSL mg/kg	MCL-based SSL mg/kg
Acephate	30560-19-1	8.7E-03	I			4.0E-03	I					1	0.1		5.6E+01	c**	2.0E+02	c*					7.7E+00	c*		1.9E-03	
Acetaldehyde	75-07-0			2.2E-06	I			9.0E-03	I	V		1		1.1E+05	1.1E+01	c**	5.3E+01	c**	1.1E+00	c**	5.6E+00	c**	2.2E+00	c**		4.5E-04	
Acetochlor	34256-82-1					2.0E-02	I					1	0.1		1.2E+03	n	1.2E+04	n					7.3E+02	n		4.0E-01	
Acetone	67-64-1					9.0E-01	I	3.1E+01	A	V		1		1.1E+05	6.1E+04	n	6.1E+05	nms	3.2E+04	n	1.4E+05	n	2.2E+04	n		4.4E+00	
Acetone Cyanohydrin	75-86-5					3.0E-03	P	6.0E-02	P	V		1		1.1E+05	2.0E+02	n	2.1E+03	n	6.3E+01	n	2.6E+02	n	5.8E+01	n		1.2E-02	
Acetonitrile	75-05-8							6.0E-02	I	V		1		1.3E+05	8.7E+02	n	3.7E+03	n	6.3E+01	n	2.6E+02	n	1.3E+02	n		2.6E-02	
Acetophenone	98-86-2					1.0E-01	I			V		1		2.3E+03	7.8E+03	ns	1.0E+05	nms					3.7E+03	n		1.1E+00	
Acrolein	107-02-8					5.0E-04	I	2.0E-05	I	V		1		2.5E+04	1.6E-01	n	6.8E-01	n	2.1E-02	n	8.8E-02	n	4.2E-02	n		8.6E-06	
Acrylamide	79-06-1	4.5E+00	I	1.3E-03	I	2.0E-04	I					1	0.1		1.1E-01	c	3.8E-01	c	1.9E-03	c	9.4E-03	c	1.5E-02	c		3.3E-06	
Acrylic Acid	79-10-7					5.0E-01	I	1.0E-03	I			1	0.1		3.0E+04	n	2.9E+05	nm	1.0E+00	n	4.4E+00	n	1.8E+04	n		3.7E+00	
Acrylonitrile	107-13-1	5.4E-01	I	6.8E-05	I	1.0E-03	H	2.0E-03	I	V		1		1.1E+04	2.4E-01	c*	1.2E+00	c*	3.6E-02	c*	1.8E-01	c*	4.5E-02	c*		9.9E-06	
Adiponitrile	111-69-3							6.0E-03	P			1	0.1		8.5E+06	nm	3.6E+07	nm	6.3E+00	n	2.6E+01	n					
Alachlor	15972-60-8	5.6E-02	C			1.0E-02	I					1	0.1		8.7E+00	c*	3.1E+01	c					1.2E+00	c	2.0E+00	6.8E-04	1.1E-03
ALAR	1596-84-5					1.5E-01	I					1	0.1		9.2E+03	n	9.2E+04	n					5.5E+03	n		1.2E+00	
Aldicarb	116-06-3					1.0E-03	I					1	0.1		6.1E+01	n	6.2E+02	n					3.7E+01	n		9.7E-03	
Aldicarb Sulfone	1646-88-4					1.0E-03	I					1	0.1		6.1E+01	n	6.2E+02	n					3.7E+01	n		8.0E-03	
Aldrin	309-00-2	1.7E+01	I	4.9E-03	I	3.0E-05	I					1	0.1		2.9E-02	c*	1.0E-01	c	5.0E-04	c	2.5E-03	c	4.0E-03	c		8.4E-04	
Ally	74223-64-6					2.5E-01	I					1	0.1		1.5E+04	n	1.5E+05	nm					9.1E+03	n		3.1E+00	
Allyl Alcohol	107-18-6					5.0E-03	I	3.0E-04	P			1	0.1		3.1E+02	n	3.1E+03	n	3.1E-01	n	1.3E+00	n	1.8E+02	n		3.7E-02	
Allyl Chloride	107-05-1							1.0E-03	I	V		1		1.5E+03	1.8E+00	n	7.7E+00	n	1.0E+00	n	4.4E+00	n	2.1E+00	n		6.8E-04	
Aluminum	7429-90-5					1.0E+00	P	5.0E-03	P			1			7.7E+04	n	9.9E+05	nm	5.2E+00	n	2.2E+01	n	3.7E+04	n		5.5E+04	
Aluminum Phosphide	20859-73-8					4.0E-04	I					1			3.1E+01	n	4.1E+02	n					1.5E+01	n			
Amdro	67485-29-4					3.0E-04	I					1	0.1		1.8E+01	n	1.8E+02	n					1.1E+01	n		1.4E+04	
Ametryn	834-12-8					9.0E-03	I					1	0.1		5.5E+02	n	5.5E+03	n					3.3E+02	n		3.6E-01	
Aminophenol, m-	591-27-5					8.0E-02	P					1	0.1		4.9E+03	n	4.9E+04	n					2.9E+03	n		1.0E+00	
Aminophenol, p-	123-30-8					2.0E-02	P					1	0.1		1.2E+03	n	1.2E+04	n					7.3E+02	n		2.5E-01	
Amitraz	33089-61-1					2.5E-03	I					1	0.1		1.5E+02	n	1.5E+03	n					9.1E+01	n		1.2E+02	
Ammonia	7664-41-7							1.0E-01	I			1			1.4E+08	nm	6.0E+08	nm	1.0E+02	n	4.4E+02	n					
Ammonium Perchlorate	7790-98-9					7.0E-04	I					1			5.5E+01	n	7.2E+02	n					2.6E+01	n			
Ammonium Sulfamate	7773-06-0					2.0E-01	I					1			1.6E+04	n	2.0E+05	nm					7.3E+03	n			
Aniline	62-53-3	5.7E-03	I			7.0E-03	P	1.0E-03	I			1	0.1		8.5E+01	c**	3.0E+02	c*	1.0E+00	n	4.4E+00	n	1.2E+01	c*		3.4E-03	
Antimony (metallic)	7440-36-0					4.0E-04	I					0.15			3.1E+01	n	4.1E+02	n					1.5E+01	n	6.0E+00	6.6E-01	2.7E-01
Antimony Pentoxide	1314-60-9					5.0E-04	H					0.15			3.9E+01	n	5.1E+02	n					1.8E+01	n			
Antimony Potassium Tartrate	11071-15-1					9.0E-04	H					0.15			7.0E+01	n	9.2E+02	n					3.3E+01	n			
Antimony Tetroxide	1332-81-6					4.0E-04	H					0.15			3.1E+01	n	4.1E+02	n					1.5E+01	n			
Antimony Trioxide	1309-64-4					4.0E-04	H	2.0E-04	I			0.15			3.1E+01	n	4.1E+02	n	2.1E-01	n	8.8E-01	n	1.5E+01	n			
Apollo	74115-24-5					1.3E-02	I					1	0.1		7.9E+02	n	8.0E+03	n					4.7E+02	n		6.1E+02	
Aramite	140-57-8	2.5E-02	I	7.1E-06	I	5.0E-02	H					1	0.1		1.9E+01	c	6.9E+01	c	3.4E-01	c	1.7E+00	c	2.7E+00	c		1.1E-01	
Arsenic, Inorganic	7440-38-2	1.5E+00	I	4.3E-03	I	3.0E-04	I	3.0E-05	C			1	0.03		3.9E-01	c*	1.6E+00	c	5.7E-04	c*	2.9E-03	c*	4.5E-02	c	1.0E+01	1.3E-03	2.9E-01
Arsine	7784-42-1							5.0E-05	I			1			7.1E+04	n	3.0E+05	nm	5.2E-02	n	2.2E-01	n					
Assure	76578-14-8					9.0E-03	I					1	0.1		5.5E+02	n	5.5E+03	n					3.3E+02	n		3.6E+00	
Asulam	3337-71-1					5.0E-02	I					1	0.1		3.1E+03	n	3.1E+04	n					1.8E+03	n		5.2E-01	
Atrazine	1912-24-9	2.3E-01	C			3.5E-02	I					1	0.1		2.1E+00	c	7.5E+00	c					2.9E-01	c	3.0E+00	1.9E-04	2.0E-03
Avermectin B1	65195-55-3					4.0E-04	I					1	0.1		2.4E+01	n	2.5E+02	n					1.5E+01	n		4.1E-02	
Azobenzene	103-33-3	1.1E-01	I	3.1E-05	I					V		1			4.9E+00	c	2.2E+01	c	7.8E-02	c	4.0E-01	c	1.2E-01	c		5.1E-04	
Barium	7440-39-3					2.0E-01	I	5.0E-04	H			0.07			1.5E+04	n	1.9E+05	nm	5.2E-01	n	2.2E+00	n	7.3E+03	n	2.0E+03	3.0E+02	8.2E+01
Baygon	114-26-1					4.0E-03	I					1	0.1		2.4E+02	n	2.5E+03	n					1.5E+02	n		4.2E-02	
Bayleton	43121-43-3					3.0E-02	I					1	0.1		1.8E+03	n	1.8E+04	n					1.1E+03	n		1.2E+01	
Baythroid	68359-37-5					2.5E-02	I					1	0.1		1.5E+03	n	1.5E+04	n					9.1E+02	n		3.3E+02	
Benefin	1861-40-1					3.0E-01	I					1	0.1		1.8E+04	n	1.8E+05	nm					1.1E+04	n		2.1E+02	
Benomyl	17804-35-2					5.0E-02	I					1	0.1		3.1E+03	n	3.1E+04	n					1.8E+03	n		2.3E+00	
Bentazon	25057-89-0					3.0E-02	I					1	0.1		1.8E+03	n	1.8E+04	n					1.1E+03	n		3.0E-01	
Benzaldehyde	100-52-7					1.0E-01	I			V		1		1.9E+03	7.8E+03	ns	1.0E+05	nms					3.7E+03	n		9.7E-01	
Benzene	71-43-2	5.5E-02	I	7.8E-06	I	4.0E-03	I	3.0E-02	I	V		1		2.0E+03	1.1E+00	c*	5.6E+00	c*	3.1E-01	c	1.6E+00	c*	4.1E-01	c	5.0E+00	2.3E-04	2.8E-03
Benzenethiol	108-98-5					1.0E-05	H			V		1		1.4E+03	7.8E-01	n	1.0E+01	n					3.7E-01	n		2.7E-04	
Benzidine	92-87-5	2.3E+02	I	6.7E-02	I	3.0E-03	I				M	1	0.1		5.0E-04	c	7.5E-03	c	1.4E-05	c	1.8E-04	c	9.4E-05	c		5.3E-07	
Benzoic Acid	65-85-0					4.0E+00	I					1	0.1		2.4E+05	nm	2.5E+06	nm					1.5E+05	n		3.3E+01	
Benzotrichloride	98-07-7	1.3E+01	I							V		1		1.6E+02	4.9E-02	c	2.2E-01	c					5.2E-03	c		1.3E-05	
Benzyl Alcohol	100-51-6					5.0E-01	P					1	0.1		3.1E+04	n	3.1E+05	nm					1.8E+04	n		4.2E+00	
Benzyl Chloride	100-44-7	1.7E-01	I			2.0E-03	P	1.0E-03	P	V		1		6.4E+01	3.8E+00	c**	1.7E+01	c**	1.0E+00	n	4.4E+00	n	4.0E-01	c**		4.9E-04	
Beryllium and compounds	7440-41-7			2.4E-03	I	2.0E-03	I	2.0E-05	I			0.007			1.6E+02	n	2.0E+03	n	1.0E-03	c*	5.1E-03	c*	7.3E+01	n	4.0E+00	5.8E+01	3.2E+00
Bidrin	141-66-2					1.0E-04	I					1	0.1		6.1E+00	n	6.2E+01	n					3.7E+00	n		3.4E-03	
Bifenox	42576-02-3					9.0E-03	P					1	0.1		5.5E+02	n	5.5E+03	n					3.3E+02	n		2.6E+00	